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IN Victoria, Edward Jess, San Diego, CA, UNITED STATES
Marquis, David Matthew, Encinitas, CA, UNITED STATES
Jones, David S., San Diego, CA, UNITED STATES
Yu, Lin, San Diego, CA, UNITED STATES
TI APL immunoreactive peptides, conjugates thereof and methods of treatment for aPL antibody-mediated pathologies
AB aPL analogs that (a) bind specifically to B cells to which an aPL epitope binds and are disclosed. Optimized analogs lack T cell epitope(s) are useful as conjugates for treating aPL antibody-mediated diseases. Conjugates comprising aPL analogs and nonimmunogenic valency platform molecules are provides as are novel nonimmunogenic valency platform molecules and linkers. Methods of preparing and identifying said analogs, methods of treatment using said analogs, methods and compositions for preparing conjugates of said analogs and diagnostic immunoassays for aPL antibodies are disclosed.

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L99 ANSWER 2 OF 71 USPATFULL on STN
IN Chen, Ching-San, Taipei, TAIWAN, PROVINCE OF CHINA
Chen, Kuan-Chung, Changhua City, TAIWAN, PROVINCE OF CHINA
Kuan, Cheng-Chun, Taipei, TAIWAN, PROVINCE OF CHINA
Lin, Ching-Yu, I-Lan, TAIWAN, PROVINCE OF CHINA
TI Biocidal protein
AB The invention relates to novel nucleic acid and protein sequences from the mung bean Vigna radiata. The nucleic acid sequence, isolated from a bruchid resistant mung bean line, encodes a thionin-like protein with insecticidal properties.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 3 OF 71 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
SO U.S. Pat. Appl. Publ., 68 pp., Cont.-in-part of U.S. Ser. No. 701,947.
CODEN: USXXCO
IN Altman, Elliot
TI Regulated expression systems for identification, screening, and directed synthesis of stabilized bioactive peptides for therapeutic use
AB An intracellular selection system allows screening for peptide bioactivity and stability. Randomized recombinant peptides are screened for bioactivity in a tightly regulated expression system, preferably derived from the wild-type lac operon. Bioactive peptides thus identified are inherently protease- and peptidase-resistant. Also provided are bioactive peptides stabilized by a stabilizing group at the N-terminus, the C-terminus, or both. The **stabilizing group** can be a small stable protein, such as the Rop protein, glutathione sulfotransferase, **thioredoxin**, **maltose binding protein**, or **glutathione reductase**, an α-helical moiety, or one or more **proline** residues. Construction and characterization of a highly regulatable expression vector, pLAC11, and its multipurpose derivs., pLAC22 and pLAC33, is described. An in vivo approach for generating novel bioactive peptides that inhibit the growth of E. coli is disclosed. Directed synthesis of stable synthetically engineered inhibitor peptides is described.

L99 ANSWER 4 OF 71 IFIPAT COPYRIGHT 2004 IFI on STN
INF Jacobs; Bertram L., Tempe, AZ, US
Rich; Alexander, Cambridge, MA, US
IN Jacobs Bertram L; Rich Alexander
TI METHOD OF INHIBITING PATHOGENICITY OF INFECTIOUS AGENTS
AB The present invention relates to methods of detecting or identifying inhibitors of a Z-DNA binding ligand to Z-DNA, methods of inhibiting the pathogenicity of an infectious agent, antiviral therapies, and compounds that inhibit complex formation between a Z-DNA binding ligand and Z-DNA.
CLMN 56 29 Figure(s).
FIG. 1A shows the amino acid sequence of E3L from an isolate of Vaccinia virus (SEQ ID NO:1)(GenBank Accession Number: S64006 Yuwen, et al. J. Virol., 195(2): 732-744, 1993), the teachings of which are incorporated herein by reference).
FIG. 1B shows the amino acid sequence of E3L from an isolate of Variola virus (SEQ ID NO:2)(GenBank Accession Number: X69198.1, the teachings of which are incorporated herein by reference).
FIG. 1C shows the amino acid sequence of E3L from an isolate of Orf virus (SEQ ID NO:3)(GenBank Accession Number: CAA10952, the teachings of which are incorporated herein by reference).
FIG. 1D shows the amino acid sequence of E3L from an isolate of Yaba-like disease virus (SEQ ID NO:4)(GenBank Accession Number: AJ293568, see also GenBank Accession Number: NC002642.1, the teachings of which are incorporated herein by reference).
FIG. 2A is a schematic representation of a surface view of Variola E3L (dark gray) in contact with Z-DNA (light gray), with side chains of E3L represented in stick format (black), and Trp195 (which contacts Z-DNA indirectly) shown in ball and stick format.
FIG. 2B is a schematic illustration of Variola or Vaccinia E3L showing residues in contact with Z-DNA, with side chains of E3L represented in stick format, and the distances between contacting residues (in Angstroms) shown, with emphasis on charged groups.
FIG. 3A is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with wild type vaccinia virus (ZE3L); vaccinia virus in which the 83 aminoterminal amino acids of the E3L protein have been deleted (del83N); vaccinia virus in which the Z-DNA binding domain Z alpha ADAR1 has been substituted for the N-terminal domain of E3L (Z alpha ADAR1); or vaccinia virus in which proline at amino acid position 192 in the Z alpha ADAR1 construct has been mutated to alanine (P192A); over time (days post infection).
FIG. 3B is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with wild type vaccinia virus (ZE3L); vaccinia virus in which the 83 aminoterminal amino acids of the E3L protein have been deleted (del83N); vaccinia virus in which the Z-DNA binding domain Z alpha ADAR1 has been substituted for the N-terminal domain of E3L (Z alpha ADAR1); or vaccinia virus in which proline at amino acid position 193 in the Z alpha ADAR1 construct has been mutated to alanine (P193A); over time (days post infection).
FIG. 4A is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with vaccinia virus in which the Z-DNA binding domain Z alpha ADAR1 has been substituted for the N-terminal domain of E3L (Z alpha ADAR1); vaccinia virus in which tyrosine at amino acid position 177 in the Z alpha ADAR1 construct has been mutated to phenylalanine (Y177F); or vaccinia virus in which tyrosine at amino acid position 177 in the Z alpha ADAR1 construct has been mutated to alanine (Y177A); over time (days post infection). Mice were infected with vaccinia viruses containing Z alpha ADAR1-E3L chimeric genes, including the indicated mutations, via intracranial injection with the indicated number of plaque-forming units (p.f.u.) of vaccinia virus constructs in 10 mu l. The mice were monitored for mortality for two weeks.
FIG. 4B is a graph of the binding activity, which is measured by the conversion of d(CG)6 DNA from a B conformation to a Z conformation (Ellipticity (mdeg) at 255 nm) by Z alpha ADAR1 protein; Z alpha ADAR1 protein in which tyrosine at amino acid position 177 has been mutated to phenylalanine (Y177F); or Z alpha ADAR1 protein in which tyrosine at amino acid position 177 has been mutated to alanine (Y177A) over time

(seconds) as assessed by circular dichroism. Conversion of DNA from the B conformation to the Z conformation occurs as the ellipticity becomes less negative and indicates binding of the protein to the Z-DNA. The ellipticity at 255 nanometers is monitored as a function of time, using 90 mu M (base pair) of d(CG)6 and 30 mu M of protein.

FIG. 5A is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with wild type vaccinia virus (WT(ZE3L)); vaccinia virus in which the Z-DNA binding domain Z alpha ADAR1 has been substituted for the Nterminal domain of E3L (Z alpha ADAR1); vaccinia virus in which glutamine at amino acid position 186 in the Z alpha ADAR1 construct has been mutated to alanine (Q186A); vaccinia virus in which glutamic acid at amino acid position 171 in the Z alpha ADAR1 construct has been mutated to alanine (E171A); or vaccinia virus in which lysine at amino acid position 169 in the Z alpha ADAR1 construct has been mutated to alanine (K169A) over time (days post infection). Mice were infected with the indicated virus via intra-cranial injection with the 100 plaqueforming units (p.f.u.) of vaccinia virus constructs in 10 mu l. The mice were monitored for mortality for 12 days.

FIG. 5B is a graph of the binding activity, which is measured by the conversion of DNA from a B conformation to a Z conformation (Ellipticity (mdeg) at 255 nm) of Z alpha ADAR1, Z alpha ADAR1 in which glutamine at amino acid position 186 has been mutated to alanine (Q186A); Z alpha ADAR1 in which glutamic acid at amino acid position 171 has been mutated to alanine (E171A); or Z alpha ADAR1 in which lysine at amino acid position 169 has been mutated to alanine (K169A) over time (seconds) as assessed by circular dichroism. Conversion of DNA from the B conformation to the Z conformation occurs as the ellipticity becomes less negative, and indicates binding of the protein to the Z-DNA.

FIG. 6A is a graph of the lethality of vaccinia virus and variants in mice (percent survival) inoculated with vaccinia virus in which the amino-terminal domain of E3L has been replaced with the Z beta domain of ADAR1 (Z beta ADAR1); or vaccinia virus in which the amino-terminal domain of E3L has been replaced with the Zp domain of ADARI and in which the isoleucine in a position that is analogous to position 177 in Z alpha has been changed to tyrosine (Z beta ADAR1I335Y) at various doses of viral inoculation (p.f.u.). Mice were infected intra-cranially with 106 p.f.u. of either a Z beta ADAR1-E3L chimeric virus, or the chimeric virus with an 1335Y mutation and survival was monitored over time.

FIG. 6B is a graph of the binding activity, which is measured by the conversion of DNA from a B conformation to a Z conformation (Ellipticity (mdeg) at 255 nm) of vaccinia virus E3L in which the amino-terminal domain of E3L has been replaced with the Z beta domain of ADAR1 (Z beta ADAR1); or vaccinia virus E3L in which the amino-terminal domain of E3L has been replaced with the Z beta domain of ADAR1 and in which the isoleucine in a position that is analogous to position 177 in Z alpha has been changed to tyrosine ((Z beta ADAR1I335Y)) over time (seconds) as assessed by circular dichroism. Conversion of DNA from the B conformation to the Z conformation occurs as the ellipticity becomes less negative, and indicates binding of the protein to the Z-DNA.

FIG. 7 is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with wild type vaccinia virus (WT(ZE3L))); vaccinia virus in which the 83 aminoterminal amino acids of the E3L protein have been deleted (E3L Delta 83N); vaccinia virus in which the human Z-DNA binding domain Z alpha ADAR has been substituted for the N-terminal domain of E3L (Z alpha ADAR1); or vaccinia virus in which the murine Z-DNA binding domain of the DLM protein has been substituted for the N-terminal domain of E3L (Z alpha DLM1) over time (days post infection). Groups of 4 to 6 C57BL/6 mice (4-6 weeks old) were infected intra-cranially with the indicated number of plaque-forming units (p.f.u.) of vaccinia virus constructs in 10 mu l. One hundred plaque-forming units were used in this experiment, and the mice were monitored for mortality for 2 weeks. Data for wild type vaccinia virus is a composite of 4 different experiments, each with 4 mice. Percent survival is plotted against days post-infection.

FIG. 8A is an amino acid sequence alignment of portions of human Z alpha of ADAR1 (SEQ ID NO: 6), murine Z alpha of DLM (SEQ ID NO: 7), E3L of

vaccinia virus (SEQ ID NO: 8), E3L of variola virus (SEQ ID NO: 9), E3L of Orf virus (SEQ ID NO: 10), E3L of Lumpyskin virus (SEQ ID NO: 11), E3L of Swinepox virus (SEQ ID NO: 12), E3L of Yaba-like disease virus (SEQ ID NO: 13), E3L of Cowpox virus (SEQ ID NO: 14), and human Z beta of ADAR1 (SEQ ID NO:15). The numbers at either end of the sequences represent the numbering of amino acid residues in their respective proteins. Amino acid residues having the hydrophobic core are indicated by triangles, and the residues that contact nucleic acids (Z-DNA) are indicated by circles. Underlined residues indicate residues important for the protein fold and for Z-DNA recognition. The secondary structure diagram at the top of the sequences (with the numbering of human Z alpha of ADAR1) indicates the predicted secondary structure of these proteins. The GenBank Accession Numbers for the various sequences are as follows: double-stranded RNA adenosine deaminase (Homo sapiens) AAB06697. 1; tumor stroma and activated macrophage protein DLM1 (Mus musculus) NP 067369; and the E3L proteins (Vaccinia virus) AAA02759; (Variola virus) NP 042088; (Orf virus) AAC08018; (lumpy skin disease virus) AAK84995; (Swinepox) NP570192; (Yaba-like disease virus) NP 073419; and (Cowpox virus) CAC42100.

FIG. 8B is a schematic representation of a co-crystal structure of the Z alpha sub-unit of ADAR1 bound to Z-DNA. The view is down the recognition helix of the protein (at left), and a number of amino acids are shown that interact with left-handed Z-DNA **stabilized** by electrostatic and van der Waals interactions. There is an edge-to-face van der Waals interaction between Guanine 4 and Tyrosine 177. Note the van der Waals interactions between **prolines** 192, 193 and the Z-DNA backbone.

FIG. 9A is a graph of the lethality of vaccinia virus or variants in mice (percent survival). Experiments analogous to those described in FIG. 4A were carried out on the E3L gene of wild type vaccinia virus. Dose response curves are shown of the lethality following intra-cranial inoculation monitored for two weeks for different doses of the virus. Results are shown for both the virus containing wild type E3L and for virus containing Y48F and Y48A mutations.

FIG. 9B is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with vaccinia virus in which the human Z-DNA binding **domain** Z alpha ADAR containing P63A or P64A mutations has been substituted for the N-terminal **domain** of E3L (P64A or P63A) or with wild type E3L virus. Mice were infected intra-cranially with the indicated doses of wild type vaccinia virus (WT(ZE3L)), or with chimeric virus containing P63A or P64A mutations and assessed for survival.

FIG. 9C is a graph of the lethality of vaccinia virus or variants in mice (percent survival) inoculated with vaccinia virus in which the human Z-DNA binding **domain** Z alpha ADAR containing P193A or P192A mutations has been substituted for the N-terminal **domain** of E3L (P193A or P192A) or with vaccinia virus in which the human Z-DNA binding **domain** Z alpha ADAR has been substituted for the N-terminal **domain** of E3L (Z alpha ADAR1). Mice were infected intra-cranially with the indicated doses of chimeric virus (Z alpha ADAR1), or chimeric virus containing P192A or P193A mutations.

FIG. 10A is a schematic representation of a reporter vector used to carry out the yeast one-hybrid assay described herein. For reporter vector construction, bait sequences of various repeats of (dC-dG) were inserted between the reporter gene (LacZ) and the URA3 gene. The orientation of the selection marker gene, URA3, created favorable (opposite or Op) conditions for Z-DNA formation. In the pLacZcOp vector, the orientation of URA3 transcription was opposite to that of the LacZ reporter gene. As shown diagrammatically with pLacZcOp, transcription of the LacZ reporter gene was activated when h Z alpha ADAR1 carrying an activation **domain** (AD) bound to Z-DNA. The Z alpha-AD hybrid fusion was expressed from an independent vector, pACT2-Z alpha .

FIG. 10B is a schematic representation of a reporter vector used to carry out the yeast one-hybrid assay described herein. For reporter vector construction, bait sequences of various repeats of (dC-dG) were inserted between the reporter gene (LacZ) and the URA3 gene. The orientations of

the selection marker gene, URA3, created unfavorable (same or Sm) conditions for Z-DNA formation. Transcriptions from both URA3 and LacZ had the same orientation in the pLacZcSm vector. The Z alpha-AD hybrid fusion was expressed from an independent vector, pACT2-Z alpha .

FIG. 11A is a graph showing activation of transcription, assessed by pgalactosidase activity as a function of the number of (dC-dG) repeats used in the yeast one-hybrid assay described herein, using pLacZcOp transfected with pACT2 or pACT2-Z alpha expression vectors encoding a Z alpha-AD hybrid fusion protein containing hZ alpha ADAR, or a control (none) containing no Z alpha protein. The Z alpha-AD fusion protein activated transcription of the LacZ reporter gene by binding to upstream Z-DNA forming bait sequences. pLacZcOp vectors were transfected into yeast either without (none) or with pACT2 or the pACT2-Z(x expression vectors containing hZ alpha ADAR1.

FIG. 11B is a histogram showing a comparison of activation of the LacZ gene by the Z alpha-AD fusion protein in different reporter vectors, pLacZcOp and pLacZcSm using various lengths of (dC-dG) repeats in the yeast one-hybrid assay described herein. The numbers above the bars represent the fold increase of the enzyme activity in pLacZcOp compared to pLacZcSm.

FIG. 12 is a histogram of the Z-DNA binding activities of Z alpha, Z beta, Za'b, Zab and Zaa using LacZ reporter gene activation and various lengths of (dC-dG) repeats. Z alpha, Z beta and Zab all came from human ADAR1. In Zaa, the Z beta **domain** of Zab was removed and Z alpha replaced it. Za'b had mutations in Z alpha of Zab. Vectors containing an activation **domain** fused to nothing (pACT2) or to various human Z alpha, Z beta or Zab constructs from human ADAR1 (pACT2-Z beta,-Z alpha,-Zab,-Zab and -Zaa) were co-transfected into yeast with pLacZcOp-(dCdG)n (where n=4, 5, 9, and 12). betagalactosidase activities were determined by a quantitative onitrophenyl-beta-D-galactose (ONPG) assay.

FIG. 13 is a histogram showing Z-DNA specific binding of Z beta or Za homologues in vivo, as assessed by measuring betagalactosidase reporter activity in yeast containing various vectors containing an activation **domain** fused to human Z beta or Z alpha constructs from human ADAR1. Vectors coding for Z alpha or its homologues in the pACT2 vector were transfected into yeast cells carrying a reporter vector, placZcOp-(dC-dG)9. All constructs were fused to an activation **domain** except pACT2 which had only an activation **domain** . The beta-galctosidase activity was determined quantitatively by o-nitrophenyl-betaD-galactose (ONPG) assay. ZE3L and Z alpha DLM were N-terminal Z alpha homologous **domains** from vaccinia virus E3L protein and mouse DLM-1, respectively. Z alpha N173A was a mutant with significantly reduced Z-DNA binding activity because of the change in the conserved Asn173 to Ala of hZ alpha ADAR.

FIG. 14A is a graph showing the results of a sensitive circular dichroism (CD) assay for Z-DNA binding specificity using the BZ equilibrium at midpoint, as described herein. The CD midpoint of the B-Z equilibrium of poly (dC-dG) was achieved by adding cobalt hexaamine. If a protein bound specifically to the Zconformation of DNA rather than the B-conformation, the equilibrium between B- and Z-conformations changed toward Z-DNA. Likewise, a B-conformation specific binding protein changed toward B-DNA. Sixty mu M of protein was added into 60 mu M (base pair) of poly (dC-dG) DNA at the midpoint of the B-Z equilibrium. The shift of B-Z equilibrium was monitored with CD at 292 nm for 3000 seconds. vZE3L (dark line) showed an apparent Z-DNA specificity by changing the CD signal to the Zconformation, while hZ beta ADAR (light line) was the opposite.

FIG. 14B shows CD spectra between 240 nm and 320 nm that were taken at 20 hours after proteins were added in the assays described in FIG. 14A. The B-Z midpoint spectrum (----) was achieved by adding cobalt hexamine around 75 mu M. Characteristics of the conformational specificity of cZE3L (dark line) and hZ(3ADAR (light line) are shown. B-DNA (open circles) and salt-induced (4 M NaCl) Z-DNA (solid circles) spectra of poly (dC-dG) were shown for comparison.

FIG. 15 shows a graph of the activation of transcription of the reporter gene LacZ, by Z alpha as assessed by measuring betagalactosidase

activity. Expression of Z alpha alone without the Gal4 activation **domain** was examined. pGNA-Z alpha expressed Z alpha with the SV40 T-antigen nuclear localization signal at the N-terminus (described herein). Experiments were performed with different pLacZcOp-(dC-dG)n reporters. hZ alpha ADAR expression increased beta-galctosidase activity significantly as stretches of dC-dG became longer.!

L99 ANSWER 5 OF 71 USPATFULL on STN
IN Frederick, Christin, Newton, MA, UNITED STATES
Saito, Haruo, Newton, MA, UNITED STATES
TI Receptor linked protein tyrosine phosphatases
AB The crystal structures of CD45 and LAR, described herein, provide a basis for kinetic and functional studies. Identification of the crystal structures of cellular molecules is important in to determine functional roles in immunity, phosphorylation events, disease initiation mechanism. The isolated crystals and methods for crystallization thereof, are also important in identifying small molecule interactions with cellular molecules for new drug discovery

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 6 OF 71 USPATFULL on STN
IN Holmes, Kathryn V., Golden, CO, UNITED STATES
Zelus, Bruce D., Lakewood, CO, UNITED STATES
Tan, Kemin, Waltham, MA, UNITED STATES
Wang, Jia-Huai, Belmont, MA, UNITED STATES
Meijers, Rob, Sommerville, MA, UNITED STATES
TI Carcinoembryonic antigen cell adhesion molecule 1 (CEACAM1) structure and uses thereof in drug identification and screening
AB Disclosed is the first crystal structure in the carcinoembryonic antigen (CEA) family, the mouse CEACAM1a[1,4], containing the N-terminal functional domain that is characterized as having a uniquely folded CC' loop. This novel feature could not be predicted based on sequence analysis alone. The structure has provided a prototypic architecture for modeling human homologues within the CEA family. These tertiary structures are used in a number of screening methods for identifying candidate molecules that have a binding affinity for the tertiary structure of the CC' loop and its vicinity. Pharmaceutical preparations that include one or more of such identified candidates may then be provided and used in treatments for certain bacterial and viral infections, certain tumors and disorders of angiogenesis or immune responses and autoimmune disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 7 OF 71 USPATFULL on STN
IN Holmes, Kathryn V., Golden, CO, UNITED STATES
Zelus, Bruce D., Lakewood, CO, UNITED STATES
Tan, Kemin, Waltham, MA, UNITED STATES
Wang, Jia-Huai, Belmont, MA, UNITED STATES
Meijers, Rob, Somerville, MA, UNITED STATES
TI Carcinoembryonic antigen cell adhesion molecule 1 (CEACAM1) structure and uses thereof in drug identification and screening
AB Disclosed are novel crystal structures of a carcinoembryonic cell adhesion antigen functional domain that is characterized as having a unique N-terminal domain structure, called a CC' loop. This tertiary structure is used in a number of screening methods for identifying candidate molecules that have a binding affinity for the tertiary structure of the CC' loop. Pharmaceutical preparations that include one or more of such identified candidate may then be provided and used in treatments for bacterial infections, dysentery, angiogenesis, immune cell mediated disease, and related conditions thereto.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 8 OF 71 USPATFULL on STN

IN Guenzler-Pukall, Volkmar, San Leandro, CA, UNITED STATES
Neff, Thomas B., Atherton, CA, UNITED STATES
Wang, Qingjian, Davis, CA, UNITED STATES
Arend, Michael P., San Mateo, CA, UNITED STATES
Flippin, Lee A., Woodside, CA, UNITED STATES
Melekhov, Alex, San Mateo, CA, UNITED STATES
TI Stabilization of hypoxia inducible factor (HIF) alpha
AB The present invention relates to methods of stabilizing the alpha subunit of hypoxia inducible factor (HIF). The invention further relates to methods of preventing, pretreating, or treating conditions associated with HIF, including ischemic and hypoxic conditions. Compounds for use in these methods are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 9 OF 71 USPATFULL on STN
IN Renner, Wolfgang A., Zurich, SWITZERLAND
Bachmann, Martin, Winterthur, SWITZERLAND
Tissot, Alain, Zurich, SWITZERLAND
Maurer, Patrick, Winterthur, SWITZERLAND
Lechner, Franziska, Zurich, SWITZERLAND
Sebbel, Peter, Zurich, SWITZERLAND
Piossek, Christine, Winterthur, SWITZERLAND
Ortmann, Rainer, Saint Louis, SWITZERLAND
Luond, Rainer, Therwil, SWITZERLAND
Staufenbiel, Matthias, Lorrach, GERMANY, FEDERAL REPUBLIC OF
Frey, Peter, Bern, SWITZERLAND
TI Molecular antigen array
AB The present invention is related to the fields of molecular biology, virology, immunology and medicine. The invention provides a composition comprising an ordered and repetitive antigen or antigenic determinant array. The invention also provides a process for producing an antigen or antigenic determinant in an ordered and repetitive array. The ordered and repetitive antigen or antigenic determinant is useful in the production of vaccines for the treatment of infectious diseases, the treatment of allergies and as a pharmaccine to prevent or cure cancer and to efficiently induce self-specific immune responses, in particular antibody responses.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 10 OF 71 USPATFULL on STN
IN Renner, Wolfgang A., Zurich, SWITZERLAND
Bachmann, Martin, Winterthur, SWITZERLAND
Tissot, Alain, Zurich, SWITZERLAND
Maurer, Patrick, Winterthur, SWITZERLAND
Lechner, Franziska, Zurich, SWITZERLAND
Sebbel, Peter, Zurich, SWITZERLAND
Piossek, Christine, Winterthur, SWITZERLAND
TI Molecular antigen array
AB The present invention is related to the fields of molecular biology, virology, immunology and medicine. The invention provides a composition comprising an ordered and repetitive antigen or antigenic determinant array. The invention also provides a process for producing an antigen or antigenic determinant in an ordered and repetitive array. The ordered and repetitive antigen or antigenic determinant is useful in the production of vaccines for the treatment of infectious diseases, the treatment of allergies and as a pharmaccine to prevent or cure cancer and to efficiently induce self-specific immune responses, in particular antibody responses.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 11 OF 71 USPATFULL on STN
IN King, Gordon E., Shoreline, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES

Xu, Jiangchun, Bellevue, WA, UNITED STATES
Secrist, Heather, Seattle, WA, UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED STATES
TI Compositions and methods for the therapy and diagnosis of colon cancer
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 12 OF 71 USPATFULL on STN
IN Tsai, Fong-Ying, Newton, MA, UNITED STATES
TI Novel SLGP protein and nucleic acid molecules and uses therefor
AB The invention provides isolated nucleic acids molecules, designated SLGP nucleic acid molecules, which encode novel GPCR family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing SLGP nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an SLGP gene has been introduced or disrupted. The invention still further provides isolated SLGP proteins, fusion proteins, antigenic peptides and anti-SLGP antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 13 OF 71 USPATFULL on STN
IN Polansky, Hanan, Rochester, NY, UNITED STATES
TI Diagnosis methods based on microcompetition for a limiting GABP complex
AB Microcompetition for GABP between a foreign polynucleotide and cellular GABP regulated genes is a risk factor associated with many chronic diseases such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present assays for the diagnosis of these chronic diseases. The assays are based on measuring the cellular copy number of the foreign polynucleotide, measuring the rate of complex formation between GABP and either the foreign polynucleotide, or a cellular GABP regulated gene, identifying modified expression of a cellular GABP regulated gene, or identifying modified activity of the gene product of a GABP regulated gene. The invention also presents other foreign polynucleotide-type assays.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 14 OF 71 USPATFULL on STN
IN Tsai, Fong-Ying, Newton, MA, UNITED STATES
TI Novel SLGP nucleic acid molecules and uses therefor
AB The invention provides isolated nucleic acids molecules, designated SLGP nucleic acid molecules, which encode novel GPCR family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing SLGP nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an SLGPgene has been introduced or disrupted. The invention still further provides isolated SLGP proteins, fusion proteins, antigenic peptides and anti-SLGP antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 15 OF 71 USPATFULL on STN
IN Benson, Darin R., Seattle, WA, UNITED STATES
Kalos, Michael D., Seattle, WA, UNITED STATES

Lodes, Michael J., Seattle, WA, UNITED STATES
Persing, David H., Redmond, WA, UNITED STATES
Hepler, William T., Seattle, WA, UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED STATES

TI Compositions and methods for the therapy and diagnosis of pancreatic cancer

AB Compositions and methods for the therapy and diagnosis of cancer, particularly pancreatic cancer, are disclosed. Illustrative compositions comprise one or more pancreatic tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly pancreatic cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 16 OF 71 USPATFULL on STN

IN Sebbel, Peter, Zurich, SWITZERLAND
Dunant, Nicolas, Zurich, SWITZERLAND
Bachmann, Martin, Winterthur, SWITZERLAND
Tissot, Alain, Zurich, SWITZERLAND
Lechner, Franziska, Zurich, SWITZERLAND
Renner, Wolfgang A., Zurich, SWITZERLAND
Hennecke, Frank, Zurich, SWITZERLAND
Nieba, Lars, Herisau, SWITZERLAND

TI Molecular antigen array

AB The invention provides compositions and processes for the production of ordered and repetitive antigen or antigenic determinant arrays. The compositions of the invention are useful for the production of vaccines for the prevention of infectious diseases, the treatment of allergies and the treatment of cancers. Various embodiments of the invention provide for a core particle that is coated with any desired antigen in a highly ordered and repetitive fashion as the result of specific interactions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 17 OF 71 USPATFULL on STN

IN Livesey, Stephen A., St. Eltham, AUSTRALIA
Campo, Anthony A. del, Houston, TX, UNITED STATES
Nag, Abhijit, Houston, TX, UNITED STATES
Nichols, Ken B., The Woodlands, TX, UNITED STATES
Griffey, Edward S., Conroe, TX, UNITED STATES
Coleman, Christopher, Houston, TX, UNITED STATES

TI Method for processing and preserving collagen-based tissues for transplantation

AB A method for processing and preserving an acellular collagen-based tissue matrix for transplantation is disclosed. The method includes the steps of processing biological tissues with a stabilizing solution to reduce procurement damage, treatment with a processing solution to remove cells, treatment with a cryoprotectant solution followed by freezing, drying, storage and rehydration under conditions that preclude functionally significant damage and reconstitution with viable cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 18 OF 71 USPATFULL on STN

IN Chen, Ching-San, Taipei, TAIWAN, PROVINCE OF CHINA
Chen, Kuan-Chung, Changhua, TAIWAN, PROVINCE OF CHINA
Kuan, Cheng-Chun, Taipei, TAIWAN, PROVINCE OF CHINA
Lin, Ching-Yu, I-Lan, TAIWAN, PROVINCE OF CHINA

TI Biocidal protein

AB The invention relates to novel nucleic acid and protein sequences from the mung bean Vigna radiata. The nucleic acid sequence, isolated from a bruchid resistant mung bean line, encodes a thionin-like protein with

insecticidal properties.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 19 OF 71 USPATFULL on STN
IN Zhu, Xiaotian, Watertown, MA, United States
TI Crystal of a kinase-ligand complex and methods of use
AB The invention relates to the three-dimensional structure of a crystal of a kinase enzyme complexed with a ligand. The three-dimensional structure of a protein kinase-ligand complex is disclosed. The invention also relates to methods of preparing such crystals. Kinase-ligand crystal structures wherein the ligand is an inhibitor molecule are useful for providing structural information that may be integrated into drug screening and drug design processes. Thus, the invention also relates to methods of using the crystal structure of kinase enzyme-ligand complexes for identifying, designing, selecting, or testing inhibitors of kinase enzymes, such inhibitors being useful as therapeutics for the treatment or modulation of i) diseases; ii) disease symptoms; or iii) the effect of other physiological events mediated by kinases; having one or more kinase enzymes involved in their pathology.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 20 OF 71 USPATFULL on STN
IN Rubenfield, Marc J., Framingham, MA, United States
Nolling, Jork, Ouincy, MA, United States
Deloughery, Craig, Medford, MA, United States
Bush, David, Somerville, MA, United States
TI Nucleic acid and amino acid sequences relating to pseudomonas aeruginosa for diagnostics and therapeutics
AB The invention provides isolated polypeptide and nucleic acid sequences derived from Pseudomonas aeruginosa that are useful in diagnosis and therapy of pathological conditions; antibodies against the polypeptides; and methods for the production of the polypeptides. The invention also provides methods for the detection, prevention and treatment of pathological conditions resulting from bacterial infection.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 21 OF 71 USPATFULL on STN
IN Allen, Martin J., Denver, CO, United States
Fang, Tsuei-Yun, Ames, IA, United States
Li, Yuxing, Ames, IA, United States
Liu, Hsuan-Liang, Ames, IA, United States
Chen, Hsiu-Mei, Taipei, TAIWAN, PROVINCE OF CHINA
Coutinho, Pedro, Angers, FRANCE
Honzatko, Richard, Ames, IA, United States
Ford, Clark, Ames, IA, United States
TI Protein engineering of glucoamylase to increase pH optimum, substrate specificity and thermostability
AB A fungal glucoamylase including a mutation pair Asn20Cys coupled with Ala27Cys forming a disulfide bond between the two members of the pair. The mutation provides increased thermal stability and reduced isomaltose formation to the enzyme. A fungal glucoamylase including a 311-314Loop mutation wherein reduced isomaltose formation is provided by the mutation is also provided. A fungal glucoamylase including a mutation Ser411Ala wherein increased pH optimum and reduced isomaltose formation is provided by the mutation is also provided. Combinations of the mutations in engineered glucoamylases are also provided as are combinations with other glucoamylase mutations that provide increased thermal stability, increased pH optimum and reduced isomaltose froamtion for cumulative improvements in the engineered glucoamylases. Also provided is a fungal glucoamylase including a mutation of Ser30Pro coupled with at least two stabilizing mutations forming a disulfide bond between the two stabilizing members. A fungal glucoamylase including a Ser30Pro/Gly137Ala/311-314 Loop is provided. A fungal glucoamylase

including a mutation Ser30Pro/Glu137Ala/Ser411Ala is also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 22 OF 71 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
IN DE MENDEZ, I
TI Screening inhibitor of proline-rich peptide and SH3 domain-comprising peptide interaction, by adding test compound to the labeled peptides, and comparing fluorescence signals in the presence and absence of compound.
AN 2003-344688 [33] WPIDS
CR 2003-344687 [33]
AB EP 1281963 A UPAB: 20030526
NOVELTY - Screening (M) candidate compound (I) for inhibiting proline-rich peptide (PRP) and SH3 domain-comprising peptide (SP) interaction comprising adding (I) and SP to PRP solution, where SP and PRP are labeled with fluorescent markers (FM1,FM2), respectively, exciting FM1 by energy source, measuring fluorescence signal (FS) at emission wavelength of FM2, and comparing FS obtained in the presence and absence of (I), is new.

DETAILED DESCRIPTION - Screening (M) a candidate compound for inhibiting the interaction between a proline-rich peptide (PRP) and a SH3 domain-comprising peptide (SP) by homogeneous time-resolved fluorescence technique, involves providing a buffer solution containing a PRP which is labeled with a first fluorescent marker (FM1) which is the first or second partner of paired fluorescent resonance energy transfer (FRET) fluorescence markers, adding the candidate compound to the solution, adding SP which is directly or indirectly labeled with a second fluorescent marker (FM2) which is the second or first partner of paired FRET fluorescence markers, submitting the mixture to a source of energy at a wavelength corresponding to the excitation wavelength of the first partner of paired FRET fluorescence markers and measuring the fluorescence signal at the emission wavelength of the second partner of paired FRET fluorescence markers, comparing the obtained fluorescence signal value with fluorescence signal value obtained in the absence of the candidate compound, to determine if the candidate compound inhibits the interaction between PRP and SH.

INDEPENDENT CLAIMS are also included for:

(1) a screening reagent (I) consisting of a PRP which is labeled with FM1, where the FM1 is the first or second partner of paired FRET fluorescence markers, or consisting of a SP which is directly or indirectly labeled with FM2, where FM2 is the second or first partner of paired FRET fluorescence markers;

(2) a kit (II) for the screening of a candidate compound for inhibiting the interaction between a PRP and a SH3 domain-comprising peptide, comprises a first screening reagent consisting of PRP which is labeled with FM1, where FM1 is the first or second partner of paired FRET fluorescence markers, and a second screening reagent consisting of SP which is directly or indirectly labeled with FM2, where FM2 is the second or first partner of paired FRET fluorescence markers;

(3) a complex (III) formed between PRP which is labeled with FM1, where FM1 is the first partner or the second partner of paired FRET fluorescence markers, and SP which is directly or indirectly labeled with FM2, where FM2 is the second partner or the first partner of paired FRET fluorescence markers;

(4) a complex (IV) formed between PRP which is labeled with FM1, where FM1 is the first or second partner of paired FRET fluorescence markers, and a candidate compound which inhibits the interaction between PRP and SP;

(5) a complex (V) formed between SP which is directly labeled with FM2, where FM2 is a second or first partner of paired FRET fluorescence markers, and a candidate compound which inhibits the interaction between PRP and SP; and

(6) use of a candidate compound (VI) selected by (M) for manufacturing a pharmaceutical composition.

ACTIVITY - Antiinflammatory; Antiarteriosclerotic; Antibacterial; Immunosuppressive; Cytostatic.

MECHANISM OF ACTION - Inhibitor the interaction between a

proline-rich peptide and a SH3 domain-comprising peptide (claimed).

No suitable data given.

USE - (M) is useful for screening a candidate compound for inhibiting the interaction between a proline-rich peptide and a SH3 domain-comprising peptide. The compound selected by (M) is useful for manufacturing a pharmaceutical composition (claimed). The compound selected by (M) is useful for selectively targeting proteins involved in a variety of intracellular signaling pathways, and for preventing or curing inflammation situations, chronic obstructive pulmonary disease (COPD), atherosclerosis, adult respiratory distress syndrome (ARDS), septic shock, cancer and general aging process.

ADVANTAGE - (M) is highly efficient, specific, sensitive and reproducible which allows for its use for screening compounds that are able to inhibit the specific binding interactions between PRP and SP which occurs in an homogeneous liquid phase without immobilizing any of the interacting protein partners. (M) does not require the use of full-length proteins nor of large polypeptides because there is no need for amino acid sequences flanking the interacting SH3 **domains** and **proline**-rich **motifs** that are used to **stabilize** binding in immunoblotting assays or alternatively that are used as linking arms bound to the reaction cuvette in real-time interaction analysis assays.

DESCRIPTION OF DRAWING(S) - The figure shows a schematic illustration of a screening method for identifying compounds which inhibit the binding between proline-rich region contained in the p22-phox protein and the SH3 domains contained in the p47-phox protein.

Dwg.1/8

L99 ANSWER 23 OF 71 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN

IN DE MENDEZ, I

TI Screening inhibitor of proline-rich peptide and SH3 domain-comprising peptide interaction, by adding test compound to the labeled peptides, and comparing fluorescence signals in the presence and absence of compound.

AN 2003-344687 [33] WPIDS

CR 2003-344688 [33]

AB EP 1281962 A UPAB: 20030813

NOVELTY - Screening (M) candidate compound (I) for inhibiting proline-rich peptide (PRP) and SH3 domain-comprising peptide (SP) interaction, comprising adding (I) and SP to PRP solution, where SP and PRP are labeled with fluorescent markers (FM1,FM2), respectively, exciting FM1 by energy source, measuring fluorescence signal (FS) at emission wavelength of FM2, and comparing FS obtained in the presence and absence of (I), is new.

DETAILED DESCRIPTION - Screening (M) a candidate compound for inhibiting the interaction between a proline-rich peptide (PRP) and a SH3 domain-comprising peptide (SP) by homogeneous time-resolved fluorescence technique, comprising:

(a) providing a buffer solution containing a PRP which is labeled with a first fluorescent marker (FM1) which is the first or second partner of paired fluorescent resonance energy transfer (FRET) fluorescence markers;

(b) adding the candidate compound to the solution;

(c) adding SP which is directly or indirectly labeled with a second fluorescent marker (FM2) which is the second or first partner of paired FRET fluorescence markers;

(d) submitting the mixture to a source of energy at a wavelength corresponding to the excitation wavelength of the first partner of paired FRET fluorescence markers and measuring the fluorescence signal at the emission wavelength of the second partner of paired FRET fluorescence markers; and

(e) comparing the obtained fluorescence signal value with fluorescence signal value obtained in the absence of the candidate compound, to determine if the candidate compound inhibits the interaction between PRP and SH.

INDEPENDENT CLAIMS are also included for:

(1) a screening reagent (I) consisting of a PRP which is labeled with FM1, where the FM1 is the first or second partner of paired FRET

fluorescence markers, or consisting of a SP which is directly or indirectly labeled with FM2, where FM2 is the second or first partner of paired FRET fluorescence markers;

(2) a kit (II) for the screening of a candidate compound for inhibiting the interaction between a PRP and a SH3 domain-comprising peptide, comprises a first screening reagent consisting of PRP which is labeled with FM1, where FM1 is the first or second partner of paired FRET fluorescence markers, and a second screening reagent consisting of SP which is directly or indirectly labeled with FM2, where FM2 is the second or first partner of paired FRET fluorescence markers;

(3) a complex (III) formed between PRP which is labeled with FM1, where FM1 is the first partner or the second partner of paired FRET fluorescence markers, and SP which is directly or indirectly labeled with FM2, where FM2 is the second partner or the first partner of paired FRET fluorescence markers;

(4) a complex (IV) formed between PRP which is labeled with FM1, where FM1 is the first or second partner of paired FRET fluorescence markers, and a candidate compound which inhibits the interaction between PRP and SP;

(5) a complex (V) formed between SP which is directly labeled with FM2, where FM2 is a second or first partner of paired FRET fluorescence markers, and a candidate compound which inhibits the interaction between PRP and SP; and

(6) use of a candidate compound (VI) selected by (M) for manufacturing a pharmaceutical composition.

ACTIVITY - Antiinflammatory; Antiarteriosclerotic; Antibacterial; Immunosuppressive; Cytostatic.

MECHANISM OF ACTION - Inhibitor the interaction between a proline-rich peptide and a SH3 domain-comprising peptide (claimed).

No biological data is given.

USE - (M) is useful for screening a candidate compound for inhibiting the interaction between a proline-rich peptide and a SH3 domain-comprising peptide. The compound selected by (M) is useful for manufacturing a pharmaceutical composition. (All claimed.) The compound selected by (M) is useful for selectively targeting proteins involved in a variety of intracellular signaling pathways, and for preventing or curing inflammation situations, chronic obstructive pulmonary disease (COPD), atherosclerosis, adult respiratory distress syndrome (ARDS), septic shock, cancer and general aging process.

ADVANTAGE - (M) is highly efficient, specific, sensitive and reproducible which allows for its use for screening compounds that are able to inhibit the specific binding interactions between PRP and SP which occurs in an homogeneous liquid phase without immobilizing any of the interacting protein partners. (M) does not require the use of full-length proteins nor of large polypeptides because there is no need for amino acid sequences flanking the interacting SH3 **domains** and **proline**-rich **motifs** that are used to **stabilize** binding in immunoblotting assays or alternatively that are used as linking arms bound to the reaction cuvette in real-time interaction analysis assays.

Dwg.0/8

L99 ANSWER 24 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN DUPLICATE

SO Journal of Biological Chemistry, (25 APR 2003), 278/17 (15406-15411), 26 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258

AU D'Angelo G.; Duplan E.; Vigne P.; Frelin C.

TI Cyclosporin A prevents the hypoxic adaptation by activating hypoxia-inducible factor-1α Pro-564 hydroxylation

AN 2003:36799880 BIOTECHNO

AB The mechanism by which hypoxia induces gene transcription involves the inhibition of hypoxia-inducible factor (HIF)-1α prolyl hydroxylase activity, which prevents von Hippel-Lindau (vHL)-dependent targeting of HIF-1α to the ubiquitin-proteasome pathway. HIF-1α is **stabilized**, translocates to the nucleus, interacts with

hypoxia-responsive elements, and promotes the activation of target genes. This report shows that cyclosporin A (CsA) interferes with the hypoxic signaling cascade in C6 glioma cells. CsA inhibits hypoxia-dependent gene transcription in a reporter gene assay and prevents the hypoxic accumulation of HIF-1α. Addition of the 530-603 C-terminal oxygen-dependent degradation (ODD) **domain** of HIF-1α to the green fluorescent protein (GFP) destabilized the protein in an oxygen-dependent manner. CsA prevented the hypoxic **stabilization** of an ODD.midldot.GFP fusion protein. An assay for 2-oxoglutarate-dependent dioxygenases was developed using a light mitochondrial kidney fraction as a source of enzyme. It uses the capacity of specific peptides to stimulate the degradation of [.sup.1.sup.4C]2-oxoglutarate. CsA stimulated the enzymatic activity in the presence of a peptide that mimicked the 557-576 sequence of HIF-1α. The enzyme promoted [.sup.3.sup.5S]vHL binding to **glutathione S-transferase** (**GST**).midldot.ODD fusion protein. This association increased in the presence of CsA. CsA effects were not observed when the **proline** residue corresponding to Pro-564 in the HIF-1α sequence was replaced by a hydroxyproline or an alanine residue. Finally, CsA increased VHL-ODD interaction during hypoxia. We conclude that CsA destabilizes HIF-1α by promoting hydroxylation of Pro-564 in the ODD **domain**. Such a mechanism may prevent hypoxic adaptation during CsA-induced nephrotoxicity and contribute to the adverse effects of this drug.

L99 ANSWER 25 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE
SO Journal of Biological Chemistry, (07 FEB 2003), 278/6 (3786-3792), 34 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258
AU Masumiya H.; Wang R.; Zhang J.; Xiao B.; Chen S.R.W.
TI Localization of the 12.6-kDa FK506-binding protein (FKBP12.6) binding site to the NH.sub.2-terminal domain of the cardiac Ca.sup.2.sup.+ release channel (ryanodine receptor)
AN 2003:36801105 BIOTECHNO
AB The 12.6-kDa FK506-binding protein (FKBP12.6) interacts with the cardiac ryanodine receptor (RyR2) and modulates its channel function. However, the molecular basis of FKBP12.6-RyR2 interaction is poorly understood. To investigate the significance of the isoleucine. **proline** (residues 2427-2428) dipeptide epitope, which is thought to form an essential part of the FKBP12.6 binding site in RyR2, we generated single and double mutants, P2428Q, I2427E/P2428A, and P2428A/L2429E, expressed them in HEK293 cells, and assessed their ability to bind **GST**-FKBP12.6 None of these mutations abolished **GST**-FKBP12.6 binding, indicating that this isoleucine-**proline motif** is unlikely to form the core of the FKBP12.6 binding site in RyR2. To systematically define the molecular determinants of FKBP12.6 binding, we constructed a series of internal and NH.sub.2- and COOH-terminal deletion mutants of RyR2 and examined the effect of these deletions on **GST**-FKBP12.6 binding. These deletion analyses revealed that the first 305 NH.sub.2terminal residues and COOH-terminal residues 1937-4967 are not essential for **GST**-FKBP12.6 binding, whereas multiple sequences within a large region between residues 305 and 1937 are required for GSTFKBP12.6 interaction. Furthermore, an NH.sub.2-terminal fragment containing the first 1937 residues is sufficient for **GST**-FKBP12.6 binding. Co-expression of overlapping NH.sub.2 and COOH-terminal fragments covering the entire sequence of RyR2 produced functional channels but did not restore **GST**-FKBP12.6 binding. These data suggest that FKBP12.6 binding is likely to be conformationdependent. Binding of FKBP12.6 to the NH.sub.2-terminal **domain** may play a role in **stabilizing** the conformation of this region.

L99 ANSWER 26 OF 71 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
SO BIOPHYSICAL CHEMISTRY, (1 AUG 2003) Vol. 105, No. 1, pp. 89-104.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM,

NETHERLANDS.
ISSN: 0301-4622.

AU Park H S; Kim C; Kang Y K (Reprint)

TI Preferred conformations of cyclic Ac-Cys-Pro-Xaa-Cys-NHMe peptides: a model for chain reversal and active site of disulfide oxidoreductase

AB The conformational study on cyclic Ac-Cys-Pro-Xaa-Cys-NHMe (Ac-CPXC-NHMe; X=Ala, Val, Leu, Aib, Gly, His, Phe, Tyr, Asn and Ser) peptides has been carried out using the Empirical Conformational Energy Program for Peptides, version 3 (ECEPP/3) force field and the hydration shell model in the unhydrated and hydrated states. This work has been undertaken to investigate structural implications of the CPXC sequence as the chain reversal for the initiation of protein folding and as the **motif** for active site of disulfide oxidoreductases. The backbone conformation DAAA is commonly the most feasible for cyclic CPXC peptides in the hydrated state, which has a type I beta-turn at the Pro-Xaa sequence. The **proline** residue and the hydrogen bond between backbones of two cystines as well as the formation of disulfide bond appear to play a role in **stabilizing** this preferred conformation of cyclic CPXC peptides. However, the distributions of backbone conformations and beta-turns may indicate that the cyclic CPXC peptide seems to exist as an ensemble of beta-turns and coiled conformations in aqueous solution. The intrinsic stability of the cyclic CPXC **motif** itself for the active conformation seems to play a role in determining electrochemical properties of disulfide oxidoreductases.

L99 ANSWER 27 OF 71 USPATFULL on STN

IN Terman, David S., Pebble Beach, CA, UNITED STATES

TI Compositions and methods for treatment of neoplastic disease

AB The present invention comprises compositions and methods for treating a tumor or neoplastic disease in a host, The methods employ conjugates comprising superantigen polypeptides, nucleic acids with other structures that preferentially bind to tumor cells and are capable of inducing apoptosis. Also provided are superantigen-glycolipid conjugates and vesicles that are loaded onto antigen presenting cells to activate both T cells and NKT cells. Cell-based vaccines comprise tumor cells engineered to express a superantigen along with glycolipids products which, when expressed, render the cells capable of eliciting an effective anti-tumor immune response in a mammal into which these cells are introduced. Included among these compositions are tumor cells, hybrid cells of tumor cells and accessory cells, preferably dendritic cells. Also provided are tumoricidal T cells and NKT cells devoid of inhitory receptors or inhibitory signaling motifs which are hyperresponsive to the the above compositions and lipid-based tumor associated antigens that can be administered for adoptive immunotherapy of cancer and infectious diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 28 OF 71 USPATFULL on STN

IN Horwath, Kathleen L., Endwell, NY, UNITED STATES
Easton, Christopher M., Ithaca, NY, UNITED STATES

TI Nucleic acid sequences encoding type III tenebrio antifreeze proteins and method for assaying activity

AB Thermal hysteresis proteins and their nucleotide sequences derived from the Tenebrionoidea Superfamily which lower the freezing point of a solution without effecting the melting point. Related methods for preparing said proteins and for providing antifreeze or recrystallization inhibition properties to a subject formulation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 29 OF 71 USPATFULL on STN

IN Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Silos-Santiago, Inmaculada, Cambridge, MA, UNITED STATES

TI 52871, a novel human G protein coupled receptor and uses thereof

AB The invention provides isolated nucleic acids molecules, designated 52871 nucleic acid molecules, which encode novel G-Protein Coupled Receptor molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 52871 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 52871 gene has been introduced or disrupted. The invention still further provides isolated 52871 proteins, fusion proteins, antigenic peptides and anti-52871 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 30 OF 71 USPATFULL on STN
IN Stolk, John A., Bothell, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Chenault, Ruth A., Seattle, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
TI Compositions and methods for the therapy and diagnosis of colon cancer
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 31 OF 71 USPATFULL on STN
IN Bachmann, Martin F., Winterhur, SWITZERLAND
Renner, Wolfgang A., Zurich, SWITZERLAND
TI Compositions for inducing self-specific anti-IgE antibodies and uses thereof
AB The invention relates to compositions for the induction of anti-IgE antibodies in order to prevent or inhibit IgE-mediated disorders. The compositions contain carriers foreign to the immunized human or animal coupled to polypeptides containing fragments of the IgE molecule. The fragment of the IgE molecule includes the constant CH1 and/or the CH4 domain of the IgE molecule. The composition is administered to humans or animals in order to induce antibodies specific for endogenous IgE antibodies. These induced anti-IgE antibodies reduce or eliminate the pool of free IgE in the serum. Since many allergic diseases are mediated by IgE, IgE-mediated disorders are ameliorated in treated mammals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 32 OF 71 USPATFULL on STN
IN Algate, Paul A., Issaquah, WA, UNITED STATES
Jones, Robert, Seattle, WA, UNITED STATES
Harlocker, Susan L., Seattle, WA, UNITED STATES
TI Compositions and methods for the therapy and diagnosis of ovarian cancer
AB Compositions and methods for the therapy and diagnosis of cancer, particularly ovarian cancer, are disclosed. Illustrative compositions comprise one or more ovarian tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly ovarian cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 33 OF 71 USPATFULL on STN
IN King, Gordon E., Shoreline, WA, UNITED STATES

Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Secrist, Heather, Seattle, WA, UNITED STATES
TI Compositions and methods for the therapy and diagnosis of colon cancer
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 34 OF 71 USPATFULL on STN
IN Yu, Yeon Gyu, Seoul, KOREA, REPUBLIC OF
Kim, Key-Sun, Seoul, KOREA, REPUBLIC OF
Jin, Bong-Suk, Seoul, KOREA, REPUBLIC OF
TI Peptides for inhibition of HIV infection
AB The present invention relates to peptides that can inhibit the infection of HIV, and more particularly, to peptides consisting of less than 30 amino acids which can introduce a helix capping motif into a peptide derived from C-terminal helical region (its 628-646.sup.th amino acid region) of gp41, an envelope glycoprotein of HIV, as well as consisting of the symmetrical bivalent peptide through the introduction of a branched amino acid, Fmoc-Lys(Fmoc)-OH at C-terminus of its peptide, and induce a more stable helical structure thus inhibiting the infection of HIV.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 35 OF 71 USPATFULL on STN
IN Holtzman, Douglas A., Jamaica Plain, MA, UNITED STATES
McCarthy, Sean A., San Diego, CA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Busfield, Samantha J., Maddington, AUSTRALIA
Pan, Yang, Bellevue, WA, UNITED STATES
White, David, Braintree, MA, UNITED STATES
Khodadoust, Mehran M., Brookline, MA, UNITED STATES
Gu, Wei, Brookline, MA, UNITED STATES
TI Novel ITALY, Lor-2, STRIFE, TRASH, BDSF, LRSG, and STMST protein and nucleic acid molecules and uses therefor
AB Novel ITALY, LOR-2, STRIFE, TRASH, BDSF, LRSG, and STMST polypeptides, proteins, and nucleic acid molecules are disclosed. In addition to isolated, full-length ITALY, LOR-2, STRIFE, TRASH, BDSF, LRSG, and STMST proteins, the invention further provides isolated ITALY, LOR-2, STRIFE, TRASH, BDSF, LRSG, and STMST fusion proteins, antigenic peptides and anti- ITALY, LOR-2, STRIFE, TRASH, BDSF, LRSG, and STMST antibodies. The invention also provides ITALY, LOR-2, STRIFE, TRASH, BDSF, LRSG, and STMST nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced and non-human transgenic animals in which an ITALY, LOR-2, STRIFE, TRASH, BDSF, LRSG, and STMST gene has been introduced or disrupted. Diagnostic, screening and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 36 OF 71 USPATFULL on STN
IN Kranz, David M., Champaign, IL, UNITED STATES
Wittrup, K. Dane, Chestnut Hill, MA, UNITED STATES
Holler, Phillip D., Champaign, IL, UNITED STATES
TI High affinity TCR proteins and methods
AB T cell receptors (TCRs) that have higher affinity for a ligand than wild type TCRs are provided. These high affinity TCRs are formed by

mutagenizing a T cell receptor protein coding sequence to generate a variegated population of mutants of the T cell receptor protein coding sequence; transforming the T cell receptor mutant coding sequence into yeast cells; inducing expression of the T cell receptor mutant coding sequence on the surface of yeast cells; and selecting those cells expressing T cell receptor mutants that have higher affinity for the peptide/MHC ligand than the wild type T cell receptor protein. The high affinity TCRs can be used in place of an antibody or single chain antibody.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 37 OF 71 USPATFULL on STN
IN Gruskin, Elliott A., Killingworth, CT, United States
Buechter, Douglas D., Wallingford, CT, United States
Zhang, Guanghui, Guilford, CT, United States
Connelly, Kevin, Los Angeles, CA, United States
TI Nucleic acids encoding extracellular matrix proteins
AB Incorporation of certain amino acid analogs into polypeptides produced by cells which do not ordinarily provide polypeptides containing such amino acid analogs is accomplished by subjecting the cells to growth media containing such amino acid analogs. The degree of incorporation can be regulated by adjusting the concentration of amino acid analogs in the media and/or by adjusting osmolality of the media. Such incorporation allows the chemical and physical characteristics of polypeptides to be altered and studied. In addition, nucleic acid and corresponding proteins including a domain from a physiologically active peptide and a domain from an extracellular matrix protein which is capable of providing a self-aggregate are provided. Human extracellular matrix proteins capable of providing a self-aggregate collagen are provided which are produced by prokaryotic cells. Preferred codon usage is employed to produce extracellular matrix proteins in prokaryotics.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 38 OF 71 USPATFULL on STN
IN Roberts, Thomas M., Cambridge, MA, United States
King, Frederick J., Brookline, MA, United States
Harris, David F., Gales Ferry, CT, United States
Hu, Erding, King of Prussia, PA, United States
Spiegelman, Bruce, Waban, MA, United States
Chan, Joanne, Brookline, MA, United States
TI Differentiation enhancing factors and uses therefor
AB The present invention relates to novel SH3 domain binding protein, referred to herein a DEF polypeptides. The DEF polypeptides comprise several motifs including a src SH3 consensus binding sequence, four ankyrin repeats, one zinc finger domain and six copies of a proline-rich tandem repeat. DEF polypeptides may function as mediators of SH3 domain-dependent signal transduction pathways and, thus may mediate multiple signaling events such as cellular gene expression, cytoskeletal architecture, protein trafficking and endocytosis, cell adhesion, migration, proliferation and differentiation. Described herein are isolated and antisense nucleic acids molecules, recombinant expression vectors, host cells and non-human transgenic animals containing an insertion or a disruption of the DEF gene. Diagnostic, screening and therapeutic methods utilizing the compositions of the invention are also provided

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 39 OF 71 USPATFULL on STN
IN Victoria, Edward Jess, San Diego, CA, United States
Marquis, David Matthew, Encinitas, CA, United States
Jones, David S., San Diego, CA, United States
Yu, Lin, San Diego, CA, United States
TI APL immunoreactive peptides, conjugates thereof and methods of treatment

for APL antibody-mediated pathologies
AB aPL analogs that (a) bind specifically to B cells to which an aPL epitope binds and are disclosed. Optimized analogs lack T cell epitope(s) are useful as conjugates for treating aPL antibody-mediated diseases. Conjugates comprising aPL analogs and nonimmunogenic valency platform molecules are provides as are novel nonimmunogenic valency platform molecules and linkers. Methods of preparing and identifying said analogs, methods of treatment using said analogs, methods and compositions for preparing conjugates of said analogs and diagnostic immunoassays for aPL antibodies are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 40 OF 71 USPATFULL on STN
IN Zonana, Jonathan, Portland, OR, United States
Ferguson, Betsy M., Portland, OR, United States
Headon, Denis, Houston, TX, United States
Overbeek, Paul, Houston, TX, United States
TI Hypohidrotic ectodermal dyplasia genes and proteins
AB The DNA and amino acid sequences are disclosed for the protein ligand (EDA1-II) and receptors (dl and DL) involved in ectodermal dysplasia. Also disclosed are variant DNA and amino acid sequences, and therapeutic applications of the ligands and receptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 41 OF 71 USPATFULL on STN DUPLICATE 4
IN Welch, William J., San Francisco, CA, United States
Brown, C. Randell, San Francisco, CA, United States
Tatzelt, Jorg, Munchen, Germany, Federal Republic of
TI Correction of genetic defects using chemical chaperones
AB A method of improving a phenotypic defect in a cell that contains a conformationally defective target protein wherein the conformational defect causes the phenotype defect, comprising contacting a first cell that expresses said conformationally defective target protein with an amount of a protein stabilizing agent that is effective to improve the conformational defect, thereby improving the phenotypic defect of the first cell in comparison with a second cell having the same conformationally defective target protein and phenotypic defect, wherein the second cell is not contacted with the protein stabilizing agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 42 OF 71 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
AU ALBERTS A S
TI New Diaphanous-related formin-autoregulatory domain containing polypeptides, for use as inhibitors of cell growth, inducers of apoptosis and anti-cancer therapeutics;
mDia2 gene transfer by vector plasmid pEFmEGFP-DAD and expression in NIH3T3 cell useful for tumor gene therapy
AN 2002-05609 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - A peptide or polypeptide (I) of no more than about 130 amino acids comprising a peptide termed Diaphanous-related formin (DRF)-autoregulatory **domain** (DAD), having a sequence (S1), or a peptide essentially consisting of S1, is new.
DETAILED DESCRIPTION - A new peptide or polypeptide (I) of no more than about 130 amino acids comprises a peptide termed Diaphanous-related formin (DRF)-autoregulatory **domain** (DAD), having a sequence (S1), or a peptide essentially consisting of S1. S1 is (GA)-(VA)-M-D-x-L-L-E-x-L-(KRQ)-X-(GA)-(SGA)-(AP), where amino acids within a set of braces are interchangeable and x means any amino acid. INDEPENDENT CLAIMS are also included for the following: (1) a fusion polypeptide (II) comprising: (a) (I), optionally a linker region, and a second polypeptide that is linked to (I), or to the linker region, which second polypeptide is not natively linked to (I); or (b) a linear

multimer of two or more repeats of monomers of (I) linked end to end, directly or with a linker present between the monomer repeats; (2) a nucleic acid molecule (III) encoding (I) or (II), and comprising: (a) a first nucleic acid sequence encoding (I); (b) optionally fused in frame with the first nucleic acid sequence, a linker nucleic acid sequence encoding a linker peptide; and (c) a second nucleic acid sequence that is linked in frame to the first nucleic acid sequence or to the linker nucleic acid sequence and that encodes a second polypeptide; (3) a nucleic acid (IIIa) that hybridizes with (III) under standard stringent hybridization conditions; (4) an expression vector (IV) comprising (III) or (IIIa), operatively linked to a promoter, and optionally, additional regulatory sequences that regulate expression of the nucleic acid in a eukaryotic cell; and (5) a cell transformed or transfected with (III), (IIIa) or (IV), in which the nucleic acid is expressed.

WIDER DISCLOSURE - The following are disclosed: (1) peptides with one or more D-amino acids substituted for one or more L-amino acids; (2) longer peptides in which the basic peptidic sequence of DAD is repeated from about 2-100 times with or without intervening spacers or linkers; and (3) compounds which retain partial peptide characteristics.

BIOTECHNOLOGY - Preferred Peptide: The C-terminus of S1 has a basic **motif** of about 5 - 12 amino acids. The sequence of (I) is present in the mDia2 protein (comprising a sequence of 1171 amino acids, given in the specification). (I) binds to the GTPase-binding **domain** (GBD) of DRF proteins. The second polypeptide in (II) is **glutathione-S-transferase** or a fluorescent protein. The multimer has a formula (F). (P-Xm)n-P (F) P = (I); X = a spacer or linker selected from 1-20C alkyl, 1-20C alkenyl, 1-20C alkynyl, 1-20C polyether containing up to 9 oxygen atoms and Glyz; z = 1 - 10; m = 0 or 1; and n = 1 - 100. Preferred Vector: (IV) is a plasmid or a viral vector. Preparation: (I) may be prepared by standard recombinant techniques.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Cell growth inhibitor; actin polymerization inducer; Arp2/3 complex activation stimulator; intramolecular binding of GBD to DAD inhibitor (claimed); gene therapy. To examine the role of the DAD **domain** in DRF function, the mDia2 DAD **domain** was fused to epidermal growth factor protein (EGFP) in a mammalian expression plasmid (pEFmEGFP-DAD) that was microinjected into NIH 3T3 cells maintained in low serum (0.1 % FCS) for 24 hours. Three hours after injection, the effects on actin reorganization and activation of SRF were assayed. Actin polymerization was observed by staining cells with fluorescent TRITC-phalloidin and SRF-regulated gene expression was monitored by staining HA13 cells for the induction of a stably transformed SRE-controlled Fos-reporter gene that contained an HA-tag by indirect immunofluorescence. The effects of EGFP-DAD were compared to similar EGFP-fusion proteins containing other mDia2 **domains**, including the GBD, FH1 and FH2 sequences. While none of the other homology **domains** had an effect on actin or SRF activity, EGFP-DAD expression strongly induced the formation of actin filaments in cells.

USE - (I), a fusion polypeptide (II) comprising (I), a nucleic acid (III) encoding (I) or (II), and a vector (IV) comprising (III), are useful for inhibiting cell growth or for killing a cell e.g. tumor cell, by apoptosis, in a live animal, by introducing (I)-(IV) into a cell in which actin polymerization and/or **stabilization** of actin fibers results in growth inhibition or cell death, so that (I) or peptide expressed by (III) or (IV) causes the actin polymerization or **stabilization**, and causes the growth inhibition of apoptosis. (I) and (II) are also useful for disrupting or inhibiting the intramolecular binding of GBD to DAD in a cell, by introducing or expressing (I) or (II) in the cell, preferably by microinjection, transfection, transduction or infection of the cell with the nucleic acid or the vector. (I) - (IV) are useful for inducing actin polymerization in a cell, and for stimulating Arp2/3 complex activation, in a cell (all claimed). (I) is useful as a research tool in molecular biology, biochemistry and medicine, and is useful as an inhibitor of cell growth, inducer of apoptosis and in

anti-cancer therapeutics. (I) is useful as a biological tool to study Rho signaling and the cytoskeletal regulation pathway. (I) - (IV) is useful for treating tumor in an animal. (III) is useful in gene therapy. (I) - (IV) are also useful for screening cells for expression of mDia e.g. mDia2 gene or its human homolog.

ADMINISTRATION - 0.01 femtogram-1 picogram/cell of (I) is administered through standard administration routes. 0.001 mg-1 g of (I) is administered through topical route.

EXAMPLE - Amino acid sequence alignments of the growing formin homology (FH) protein superfamily delineated the **proline**-rich FH1 and FH2 regions of homology. Comparative alignment of only the Diaphanous-related formins (DRF) sub-family yielded a conserved **domain** in the C-termini. The consensus sequence (GA)-(VA)-M-D-x-L-L-E-x-L-(KRQ)-X-(GA)-(SGA)-(AP), was designated the DRF-autoregulatory **domain** or DAD. There was also a conserved region of basic residues several residues towards the C-terminal. The DAD **domain** was found in all the three mouse/human DRFs, budding yeast Saccharomyces cerevisiae Bni1p and Emercella nidulans SepA proteins, the exception appeared to be Bnr1p. (70 pages)

L99 ANSWER 43 OF 71 USPATFULL on STN
IN Rose, Larry M., Carmichael, CA, United States
Meares, Claude F., Davis, CA, United States
O'Donnell, Robert T., Sacramento, CA, United States
TI Antigenic epitopes with Lym-1 reactivity and uses thereof
AB This invention provides novel peptide epitopes recognized by the non-Hodgkin's B cell lymphoma reactive Lym-1 antibody. These novel peptide epitopes are capable of generating antibodies directed against Lym-1 peptide epitope expressing B-NHL cells. This invention is also directed to the treatment of B-NHL.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 44 OF 71 USPATFULL on STN
IN Hook, Magnus, Houston, TX, United States
Patti, Joseph M., Missouri City, TX, United States
House-Pompeo, Karen, Valdosta, GA, United States
Sthanam, Narayana, Vestavia, AL, United States
Symersky, Jindrich, Birmingham, AL, United States
TI Collagen binding protein compositions and methods of use
AB Disclosed are the cna gene and cna-derived nucleic acid segments from Staphylococcus aureus, and DNA segments encoding cna from related bacteria. Also disclosed are Col binding protein (CBP) compositions and methods of use. The CBP protein and antigenic epitopes derived therefrom are contemplated for use in the treatment of pathological infections, and in particular, for use in the prevention of bacterial adhesion to Col. DNA segments encoding these proteins and anti-(Col binding protein) antibodies will also be of use in various screening, diagnostic and therapeutic applications including active and passive immunization and methods for the prevention of bacterial colonization in an animal such as a human. These DNA segments and the peptides derived therefrom are contemplated for use in the preparation of vaccines and, also, for use as carrier proteins in vaccine formulations, and in the formulation of compositions for use in the prevention of S. aureus infection.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 45 OF 71 USPATFULL on STN
IN Welch, William J., San Francisco, CA, United States
Brown, C. Randell, Hershey, PA, United States
Tatzelt, Jorg, Munchen, Germany, Federal Republic of
TI Correction of genetic defects using chemical chaperones
AB A method of improving a phenotypic defect in a cell that contains a conformationally defective target protein wherein the conformational defect causes the phenotype defect, comprising contacting a first cell that expresses said confonnationally defective target protein with an

amount of a protein stabilizing agent that is effective to improve the conformational defect, thereby improving the phenotypic defect of the first cell in comparison with a second cell having the same conformationally defective target protein and phenotypic defect, wherein the second cell is not contacted with the protein stabilizing agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 46 OF 71 USPATFULL on STN
IN Rose, Larry M., Carmichael, CA, United States
Meares, Claude F., Davis, CA, United States
O'Donnell, Robert T., Sacramento, CA, United States
TI Antigenic epitopes with LYM-1 reactivity and uses thereof
AB This invention provides novel peptide epitopes recognized by the non-Hodgkin's B cell lymphoma reactive Lym-1 antibody. These novel peptide epitopes are capable of generating antibodies directed against Lym-1 peptide epitope expressing B-NHL cells. This invention is also directed to the treatment of B-NHL.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 47 OF 71 USPATFULL on STN
IN Victoria, Edward Jess, San Diego, CA, United States
Marquis, David Matthew, Encinitas, CA, United States
Jones, David S., San Diego, CA, United States
Yu, Lin, San Diego, CA, United States
TI aPL immunoreactive peptides, conjugates thereof and methods of treatment for aPL antibody-mediated pathologies
AB aPL analogs that (a) bind specifically to B cells to which an aPL epitope binds and are disclosed. Optimized analogs lack T cell epitope(s) are useful as conjugates for treating aPL antibody-mediated diseases. Methods of preparing and identifying said analogs, methods of treatment using said analogs, methods and compositions for preparing conjugates of said analogs and diagnostic immunoassays for aPL antibodies are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 48 OF 71 USPATFULL on STN
IN Levinson, Douglas Adam, Sherborn, MA, United States
TI Compositions and methods for the treatment and diagnosis of immune disorders
AB The present invention relates to methods and compositions for the treatment and diagnosis of immune disorders, especially T helper lymphocyte-related disorders. For example, genes which are differentially expressed within and among T helper (TH) cells and TH cell subpopulations, which include, but are not limited to TH0, TH1 and TH2 cell subpopulations are identified. Genes are also identified via the ability of their gene products to interact with gene products involved in the differentiation, maintenance and effector function of such TH cells and TH cell subpopulations. The genes identified can be used diagnostically or as targets for therapeutic intervention. In this regard, the present invention provides methods for the identification and therapeutic use of compounds as treatments of immune disorders, especially TH cell subpopulation-related disorders. Additionally, methods are provided for the diagnostic evaluation and prognosis of TH cell subpopulation-related disorders, for the identification of subjects exhibiting a predisposition to such conditions, for monitoring patients undergoing clinical evaluation for the treatment of such disorders, and for monitoring the efficacy of compounds used in clinical trials.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 49 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE
SO Journal of Biological Chemistry, (28 SEP 2001), 276/39 (36174-36182), 65

reference(s)
CODEN: JBCHA3 ISSN: 0021-9258

AU Gelkop S.; Babichev Y.; Isakov N.

TI T Cell Activation Induces Direct Binding of the Crk Adapter Protein to the Regulatory Subunit of Phosphatidylinositol 3-Kinase (p85) via a Complex Mechanism Involving the Cbl Protein

AN 2001:37384221 BIOTECHNO

AB The Crk adapter proteins are assumed to play a role in T lymphocyte activation because of their induced association with tyrosine-phosphorylated proteins, such as ZAP-70 and Cbl, and with the phosphatidylinositol 3-kinase regulatory subunit, p85, following engagement of the T cell antigen receptor. Although the exact mechanism of interaction between these molecules has not been fully defined, it has been generally accepted that Crk, ZAP-70, and p85 interact with tyrosine-phosphorylated Cbl, which serves as a major scaffold protein in activated T lymphocytes. Our present results demonstrate a cell activation-dependent reciprocal co-immunoprecipitation of CrkII and p85 from lysates of Jurkat T cells and a direct binding of CrkII to p85 in an overlay assay. The use of bead-immobilized **GST** fusion proteins indicated a complex mechanism of interaction between CrkII and p85 involving two distinct and mutually independent regions in each molecule. A relatively high affinity binding of the CrkII-SH3(N) **domain** to p85 and the p85-**proline**-B cell receptor-**proline** (PBP) region to CrkII was observed in lysates of either resting or activated T cells. Direct physical interaction between the CrkII-SH3(N) and the p85-PBP **domain** was demonstrated using recombinant fusion proteins and was further substantiated by binding competition studies. In addition, immobilized fusion proteins possessing the CrkII-SH2 and p85-SH3 **domains** were found to pull down p85 and CrkII, respectively, but only from lysates of activated T cells. Nevertheless, the **GST**-CrkII-SH2 fusion protein was unable to mediate direct association with p85 from lysates of either resting or activated T cells. Our results support a model in which T cell activation dependent conformational changes in CrkII and/or p85 promote an initial director indirect low affinity interaction between the two molecules, which is then **stabilized** by a secondary high affinity interaction mediated by direct binding of the CrkII-SH3(N) to the p85-PBP **domain**.

L99 ANSWER 50 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN DUPLICATE

SO Journal of Biological Chemistry, (25 JAN 2001), 276/21 (17653-17662), 58 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258

AU Hauck C.R.; Hunter T.; Schlaepfer D.D.

TI The v-Src SH3 Domain Facilitates a Cell Adhesion-independent Association with Focal Adhesion Kinase

AN 2001:37411316 BIOTECHNO

AB Integrins facilitate cell attachment to the extracellular matrix, and these interactions generate cell survival, proliferation, and motility signals. Integrin signals are relayed in part by focal adhesion kinase (FAK) activation and the formation of a transient signaling complex initiated by Src homology 2 (SH2)-dependent binding of Src family protein-tyrosine kinases to the FAK Tyr-397 autophosphorylation site. Here we show that in viral Src (v-Src)-transformed NIH3T3 fibroblasts, an adhesion-in-dependent FAK-Src signaling complex occurs. Co-expression studies in human 293T cells showed that v-Src could associate with and phosphorylate a Phe-397 FAK mutant at Tyr-925 promoting Grb2 binding to FAK in suspended cells. In vitro, **glutathione S-transferase** fusion proteins of the v-Src SH3 but not c-Src SH3 **domain** bound to FAK in lysates of NIH3T3 fibroblasts. The v-Src SH3-binding sites were mapped to known **proline**-X-X-**proline** (PXXP) SH3-binding **motifs** in the FAK N- (residues 371-377) and C-terminal **domains** (residues 712-718 and 871-882) by in vitro pull-down assays, and these sites are composed of a PXXPXXΦ (where Φ is a hydrophobic residue) v-Src SH3 binding

consensus. Sequence comparisons show that residues in the RT loop region of the c-Src and v-Src SH3 **domains** differ. Substitution of c-Src RT loop residues (Arg-97 and Thr-98) for those found in the v-Src SH3 **domain** (Trp-97 and Ile-98) enhanced the binding of distinct NIH3T3 cellular proteins to a **glutathione S-transferase** fusion protein of the c-Src (Trp-97 + Ile-98) SH3 **domain**. FAK was identified as a c-Src (Trp-97 + Ile-98) SH3 **domain** target in fibroblasts, and co-expression studies in 293T cells showed that full-length c-Src (Trp-97 + Ile-98) could associate in vivo with Phe-397 FAK in an SH2-independent manner. These studies establish a functional role for the v-Src SH3 **domain** in **stabilizing** an adhesion-independent signaling complex with FAK.

L99 ANSWER 51 OF 71 CAPLUS COPYRIGHT 2004 ACS on STN
SO PCT Int. Appl., 135 pp.
CODEN: PIXXD2
IN Altman, Elliot
TI Methods of stabilizing small peptides against proteolysis and screening them for biological activity
AB An intracellular selection system allows concurrent screening for peptide bioactivity and stability. Randomized recombinant peptides are screened for bioactivity in a tightly regulated expression system, preferably derived from the wild-type lac operon. Bioactive peptides thus identified are inherently protease- and peptidase-resistant. Also provided are bioactive peptides stabilized by a stabilizing group at either the N-terminus, the C-terminus, or both. The **stabilizing group** can take the form of a small stable protein, such as the Rop protein, glutathione sulfotransferase, **thioredoxin**, **maltose binding protein**, or **glutathione reductase**, or one or more **proline** residues.

L99 ANSWER 52 OF 71 USPATFULL on STN
IN Giordano, Antonio, Philadelphia, PA, United States
TI Human cyclin-dependent kinase-like proteins and methods of using the same
AB Substantially pure human cyclin-dependant kinase-like proteins PITALRE and PISSLRE and isolated proteinc complexes that comprise PITALRE or PISSLRE are disclosed. Isolated nucleic acid molecule that encodes PITALRE or PISSLRE, or a fragment thereof; recombinant expression vectors that comprise nucleic acid sequence that encode PITALRE or PISSLRE; and host cells that comprise such recombinant expression vectors are disclosed. Oligonucleotide molecules that consist of a nucleotide squence complimentary to a portion of the nucleotide sequence that encodes PITALRE or PISSLRE are disclosed. Antibodies which bind to epitopes on PITALRE or PISSLRE are disclosed. Nucleic acid molecules that comprise a nucleotide sequence that encodes phosphorylation deficient PITALRE or a phosphorylation deficient PISSLRE; recombinant vectors and pharmaceutical compositions that comprise such nucleotide sequences are also disclosed. Methods of identifying compounds which inhibit PITALRE activity are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 53 OF 71 USPATFULL on STN
IN Bandman, Olga, Mountain View, CA, United States
Lal, Preeti, Sunnyvale, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
TI Human pinin splice variant
AB The invention provides a human pinin splice variant (PNIN) and polynucleotides which identify and encode PNIN. The invention also provides expression vectors, host cells, agonists, antibodies and antagonists. The invention also provides methods for treating disorders associated with expression of PNIN.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 54 OF 71 USPATFULL on STN
IN Cano, Carlos Antonio Durate, Habana, Cuba
Nieto, Enrique Gerardo Guillen, Habana, Cuba
Acosta, Anabel Alvarez, Habana, Cuba
Munoz, Luis Emilio Carpio, Sancti Spiritus, Cuba
Vazquez, Diogenes Quintana, Pinar del Rio, Cuba
Rodriquez, Carmen Elena Gomez, Habana, Cuba
de la Caridid Siva Rodriguez, Recardo, Habana, Cuba
Galvez, Consuelo Nazabal, Habana, Cuba
Angulo, Maria De Jesus Leal, Habana, Cuba
Dunn, Alejandro Miguel Martin, Habana, Cuba
TI System for the expression of heterologous antigens as fusion proteins
AB The present invention relates to biotechnology and genetic engineering, particularly the expression of proteins of viral origin in microorganisms through their fusion, by applying the recombinant DNA technology, to bacterial peptides. The present invention provides an efficient process for the expression in Escherichia coli of heterologous proteins as fusion polypeptides with a view to obtaining them with a high degree of purity, in commercially useful amounts, and in an appropriate form for their inclusion in vaccine preparations intended to human use. To this effect, what is essentially used is a stabilizing sequence derived from the first 47 amino acids of the antigen P64k of Neisseria meningitidis B:4:P1.15. In particular, use is made of a recombinant plasmid containing said sequence, under the control of the tryptophane promotor of E. coli and of the terminator of the transcription of the phage T4, including restriction sites which provide for the cloning in phase of DNA fragments coding for polypeptides of interest. The process of the invention is applicable to the pharmaceutical industry, for the development of diagnostic systems, vaccine preparations, and in any situation where it is required to obtain high amounts of heterologous proteins as fusion polypeptides in E. coli.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 55 OF 71 USPATFULL on STN
IN Natesan, Sridaran, Chestnut Hill, MA, United States
TI Transcriptional activators, and compositions and uses related thereto
AB The present invention relates to chimeric transcriptional activators.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 56 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE
SO Experimental Cell Research, (15 MAR 2000), 255/2 (135-143), 56 reference(s)
CODEN: ECREAL ISSN: 0014-4827
AU Xu W.; Gong L.; Haddad M.M.; Bischof O.; Campisi J.; Yeh E.T.H.; Medrano E.E.
TI Regulation of microphthalmia-associated transcription factor MITF protein levels by association with the ubiquitin-conjugating enzyme hUBC9
AN 2000:30143848 BIOTECHNO
AB The basic helix-loop-helix/leucine zipper (bHLH/ZIP) microphthalmia-associated transcription factor (MITF) regulates transcription of genes encoding enzymes essential for melanin biosynthesis in melanocytes and retinal pigmented epithelial cells. To determine how MITF activity is regulated, we used the yeast two-hybrid system to identify proteins expressed by human melanoma cells that interact with MITF. The majority of clones that showed positive interaction with a 158-amino-acid region of MITF containing the bHLH/ZIP **domain** (aa 168-325) encoded the ubiquitin conjugating enzyme hUBC9. The association of MITF with hUBC9 was further confirmed by an in vitro **GST** pull-down assay.
Although hUBC9 is known to interact preferentially with SENTRIN/SUMO1, in vitro transcription/translation analysis demonstrated greater association of MITF with ubiquitin than with SENTRIN. Importantly, cotransfection of

MITF and hUBC9 expression vectors resulted in MITF protein degradation. MITF protein was **stabilized** by the proteasome inhibitor MG132, indicating the role of the ubiquitin-proteasome system in MITF degradation. Serine 73, which is located in a region rich in **proline**, glutamic acid, serine, and threonine (PEST), regulates MITF protein stability, since a serine to alanine mutation prevented hUBC9-mediated MITF (S73A) degradation. Furthermore, we identified lysine 201 as a potential ubiquitination site. A lysine to arginine mutation abolished MITF (K201R) degradation by hUBC9 in vivo. Our experiments indicate that by targeting MITF for proteasome degradation, hUBC9 is a critical regulator of melanocyte differentiation. (C) 2000 Academic Press.

L99 ANSWER 57 OF 71 USPATFULL on STN
IN Welch, William J., 48 Fountain, San Francisco, CA, United States 94114
Brown, C. Randell, 1470 9th Ave. #12, San Francisco, CA, United States 94122
Tatzelt, Jorg, 740 Parnassus, San Francisco, CA, United States 94122
TI Correction of genetic defects using chemical chaperones
AB The present invention provides methods of improving phenotypic defects that are caused by conformationally defective target proteins. The methods of the invention comprise exposing a cell that expresses a conformationally defective target protein with an amount of a protein stabilizing agent that is effective to improve the phenotypic defect. Nonlimiting examples of protein stabilizing agents include dimethylsulfoxide (DMSO), deuterated water, trimethylamine N-oxide (TMAO). Nonlimiting examples of defective target proteins to be treated include the cystic fibrosis transmembrane conductance regulator (CFTR) protein and prion proteins. In one embodiment, the invention provides methods for detecting protein stabilizing agents. In another embodiment, the invention provides methods for detecting cells and pathological conditions caused by improper folding and protein processing.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 58 OF 71 USPATFULL on STN
IN Hanafusa, Hidesaburo, New York, NY, United States
Knudsen, Beatrice S., New York, NY, United States
Feller, Stephan M., New York, NY, United States
Kuriyan, John, New York, NY, United States
Wu, Xiaodong, New York, NY, United States
Zheng, Jie, New York, NY, United States
Cowburn, David, Westfield, NJ, United States
TI Peptides specific for the first Crk-SH3 domain
AB The present invention relates to regulation and control of cellular processes by SH3-domain binding proteins and peptides. In particular, the invention provides a consensus sequence of a peptide that shows high specificity and affinity for the first SH3 domain of cellular Crk. In specific examples, a number of peptides that contain the consensus are shown to bind c-Crk specifically. The molecular basis for this specificity is examined by crystallography.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 59 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE
SO Protein Science, (1999), 8/1 (96-105), 40 reference(s)
CODEN: PRCIEI ISSN: 0961-8368
AU Charbonnier J.-B.; Belin P.; Moutiez M.; Stura E.A.; Quemeneur E.
TI On the role of the cis-proline residue in the active site of DsbA
AN 1999:29035523 BIOTECHNO
AB In addition to the Cys-Xaa-Xaa-Cys **motif** at position 30-33, DsbA, the essential catalyst for disulfide bond formation in the bacterial periplasm shares with other oxidoreductases of the **thioredoxin** family a cis-**proline** in proximity of the active site residues. In the variant DsbA(p151A), this residue has been

changed to an alanine, an almost isosteric residue which is not disposed to adopt the cis conformation. The substitution strongly destabilized the structure of DsbA, as determined by the decrease in the free energy of folding. The pK(a) of the thiol of Cys30 was only marginally decreased. Although in vivo the variant appeared to be correctly oxidized, it exhibited an activity less than half that of the wild-type enzyme with respect to the folding of alkaline phosphatase, used as a reporter of the disulfide bond formation in the periplasm. DsbA(P151A) crystallized in a different crystal form from the wild-type protein, in space **group** P2.sub.1 with six molecules in the asymmetric unit. Its X-ray structure was determined to 2.8 Å resolution. The most significant conformational changes occurred at the active site. The loop 149-152 adopted a new backbone conformation with Ala 151 in a trans conformation. This rearrangement resulted in the loss of van der Waals interactions between this loop and the disulfide bond. His32 from the Cys-Xaa-Xaa-Cys sequence presented in four out of six molecules in the asymmetric unit a gauche- conformation not observed in the wild-type protein. The X-ray structure and folding studies on DsbA(P151A) were consistent with the cis-**proline** playing a major role in the **stabilization** of the protein. A role for the positioning of the substrate is discussed. These important properties for the enzyme function might explain the conservation of this residue in DsbA and related proteins possessing the **thioredoxin** fold.

L99 ANSWER 60 OF 71 USPATFULL on STN
IN Gruskin, Elliott A., 23 Beech Tree Ridge, Killingworth, CT, United States 06419
Buechter, Douglas D., 51 Pierson Dr., Wallingford, CT, United States 06492
Zhang, Guanghui, 975 Little Meadow Rd., Guilford, CT, United States 06473
Connolly, Kevin, 4024 La Salle Ave., Culver City, CA, United States 90232
TI Amino acid modified polypeptides
AB Incorporation of certain amino acid analogs into polypeptides produced by cells which do not ordinarily provide polypeptides containing such amino acid analogs is accomplished by subjecting the cells to growth media containing such amino acid analogs. The degree of incorporation can be regulated by adjusting the concentration of amino acid analogs in the media and/or by adjusting osmolality of the media. Such incorporation allows the chemical and physical characteristics of polypeptides to be altered and studied.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 61 OF 71 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 9
SO Biochemistry, (14 Jul 1998) 37/28 (10286-10297).
Refs: 44
ISSN: 0006-2960 CODEN: BICHAW
AU Georgescu R.E.; Li J.-H.; Goldberg M.E.; Tasayco M.L.; Chaffotte A.F.
TI Proline isomerization-independent accumulation of an early intermediate and heterogeneity of the folding pathways of a mixed α/β protein, Escherichia coli **thioredoxin**.
AB Oxidized Escherichia coli **thioredoxin** (Trx) is a small protein of 108 residues with one disulfide bond (C32-C35 essentially involved in the activity) and no prosthetic moieties, which folds into a structural **motif** containing a central twisted β-sheet flanked by helices that is found in many larger proteins. The kinetics of refolding of Trx in vitro have been investigated using a newly developed active site titration assay and continuous or stopped-flow (SF) methods in conjunction with circular dichroism (CD) and fluorescence (Fl) spectroscopy. These studies revealed the presence of early folding intermediates with 'molten globule or pre-molten globule' characteristics. Measurements of the ellipticity at 222 nm indicated that about 68% of the total change associated with refolding occurred during the dead time (4 ms) of the stopped-flow

instrument, suggesting the formation of substantial secondary structure. The reconstruction of the far-UV CD spectrum of the burst intermediate using combined continuous and stopped- flow methods showed the formation of a defined secondary structure that contains more β-structure than the native state. Kinetic measurements using SF far-UV CD and F1 over a wide range (0.087-6 M) of GuHCl concentrations at two temperatures (6 and 20 °C) demonstrated that the population formed during the 4 ms dead time contained multiple species that are **stabilized** mainly by hydrophobic interactions and undergo further folding along alternative pathways. One of these species leads directly and rapidly to the native state as demonstrated by active site titration, while the two others fold into a fourth intermediate that is slowly converted to the native protein. Double-jump experiments suggest that the heterogeneity in folding behavior results from **proline** isomerizations occurring in the unfolded state. Conversely, the accumulation of the burst intermediate does not depend on **proline** isomerizations.

L99 ANSWER 62 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE
SO Biochemical and Biophysical Research Communications, (19 AUG 1998), 249/2 (537-541), 23 reference(s)
CODEN: BBRCA0 ISSN: 0006-291X
AU Graham L.J.; Stoica B.A.; Shapiro M.; DeBell K.E.; Rellahan B.; Laborda J.; Bonvini E.
TI Sequences surrounding the Src-homology 3 domain of phospholipase Cγ-1 increase the domain's association with Cbl
AN 1998:28431789 BIOTECHNO
AB SH3 **domains** are protein modules that interact with **proline**-rich polypeptide fragments. Cbl is an adapter-like protein known to interact with several SH3 **domains**, including the PLCγ1 SH3 **domain** and the Grb2 amino terminal SH3 **domain**. Here we explore whether sequences surrounding the PLCγ1 SH3 **domain** core sequence (aa.796-851) can affect the binding to Cbl, a target used as a prototypical ligand. Consistent with previous reports, our results demonstrated a weak binding of Cbl to **GST** fusion proteins that strictly encompass the structural core of the PLCγ1 SH3 **domain** but a high-avidity binding to the Grb2 amino-terminal SH3 **domain**. Inclusion of amino acids immediately flanking the PLCγ1 SH3 core **domain**, however, substantially increased binding of Cbl to a level comparable to that of the Grb2 amino-terminal SH3 **domain**. The interaction of this extended PLCγ1 SH3 **domain** fusion protein with Cbl was shown to depend entirely upon the interaction of the **domain** with a **proline**-rich **motif** in Cbl, ruling out the possibility that amino acids adjacent to the core SH3 **domain** of PLCγ1 provide independent Cbl binding. These data suggest that sequences surrounding the SH3 **domain** of PLCγ1 may contribute to or **stabilize** the association of the **domain** with the target protein, thus increasing its binding efficiency.

L99 ANSWER 63 OF 71 USPATFULL on STN
IN Giordano, Antonio, Philadelphia, PA, United States
TI Human cyclin-dependent kinase-like proteins and methods of using the same
AB Substantially pure human cyclin-dependent kinase-like proteins PITALRE and PISSLRE and isolated protein complexes that comprise PITALRE or PISSLRE are disclosed. Isolated nucleic acid molecule that encode PITALRE or PISSLRE, or a fragment thereof; recombinant expression vectors that comprise nucleic acid sequences that encode PITALRE or PISSLRE; and host cells that comprise such recombinant expression vectors are disclosed. Oligonucleotide molecules that consist of a nucleotide sequence complementary to a portion of the nucleotide sequence that encodes PITALRE or PISSLRE are disclosed. Antibodies which bind to epitopes on PITALRE or PISSLRE are disclosed. Nucleic acid molecules that comprise a nucleotide sequence that encodes

phosphorylation deficient PITALRE or a phosphorylation deficient PISSLRE; recombinant vectors and pharmaceutical compositions that comprise such nucleotide sequences are also disclosed. Methods of identifying compounds which inhibit PITALRE activity are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L99 ANSWER 64 OF 71 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE
SO Oncogene, (1997), 14/17 (2019-2024), 26 reference(s)
CODEN: ONCNES ISSN: 0950-9232
AU Dombrosky-Ferlan P.M.; Corey S.J.
TI Yeast two-hybrid in vivo association of the Src kinase Lyn with the protooncogene product Cbl but not with the p85 subunit of PI 3-kinase
AN 1997:27237004 BIOTECHNO
AB Ligand binding of multi-chain antigen receptors and hematopoietin/cytokine receptors results in rapid activation of protein tyrosine kinase (PTK)-dependent signalling molecules such as phosphatidylinositol 3-kinase (PI 3-kinase). Co-precipitation studies have shown that Src-related PTK, such as Lyn, associates with the p85 regulatory subunit of PI 3-kinase via SH2 and SH3 **domain** binding with their cognate ligands. More recent studies have shown that the proto-oncogene product Cbl co-precipitates with p85 following engagement of cytokine and antigen receptors. As opposed to in vitro co-precipitation studies, the yeast two-hybrid screen reveals in vivo protein-protein interactions. Using the yeast two-hybrid screen, we demonstrate an ill vivo association of Lyn's SH3 and SH2 **domains** with the **proline**-rich **domain** of Cbl. Lyn's SH3 and SH2 **domains** do not interact with p85 in the yeast two-hybrid screen, as could be predicted from **glutathione**-**S**-**transferase** (**GST**) fusion protein pull-down or co-immunoprecipitation studies from whole cell lysates. However, the SH3 **domain** of p85 interacts with the **proline**-rich **domain** of Cbl. When yeast were transformed with catalytic Lyn, an interaction between p85's SH2 **domain** and Cbl occurred. From the data, we propose the following three step process of PI 3-kinase activation: (1) complexes of Lyn-Cbl and Cbl-p85 exist without ligand stimulation, (2) upon ligand binding, Lyn becomes active and phosphorylates Cbl, and (3) Cbl's tyrosine phosphorylated residue serves as a docking site for the SH2 **domains** of p85 - thereby **stabilizing** the complex and activating PI 3-kinase. The yeast two-hybrid system can be used to dissect the precise mechanisms of ill vivo protein-protein interactions, including those between phosphotyrosine and SH2-containing proteins.

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AU Principal Investigator: MUNDEL, PETER
TI CELL AND MOLECULAR BIOLOGICAL ANALYSIS OF SYNAPTOPODIN
SUM DESCRIPTION: Podocytes are highly specialized cells that play a central role in the physiology and pathology of the kidney glomerulus. Podocytes consist of cell body, major processes and foot processes. Podocyte foot processes contain a complete actin-based contractile apparatus. Synaptopodin is a **proline**-rich, actin-associated protein of podocyte foot processes and telencephalic dendrites without significant homology to any known protein. In brain and kidney, in vivo and in vitro, synaptopodin gene expression is differentiation-dependent; in podocytes, synaptopodin expression coincides with growth arrest and process formation. The C-terminal half of synaptopodin associates with actin filaments and shows about 45 percent homology with myopodin, the second gene family member that we have cloned from skeletal and heart muscle. Myopodin colocalizes with a-actinin at the Zdisc of skeletal and heart muscle sarcomer. Similarly, synaptopodin colocalizes with a-actinin cultured podocytes and in transfected NIH3T3 fibroblasts. Therefore synaptopodin may be involved in the organization or regifiation of a Z-disc equivalent in podocytes. Mice lacking synaptopodin are viable and do not show an overt phenotype in a mixed 1 29/C57 background. However

they show a down regulation of paxillin and a-actinin in podocytes and desmin, an early marker of podocyte injury, is induced in podocytes of synpo-/- mice. Moreover, the adaptation of synaptopodin deficient mice to glomerular stress is reduced resulting in increased levels of proteinuria and albuminuria. Based upon these findings we hypothesize that synaptopodin functions as a key protein in the organization/regulation of the podocyte foot processes actin cytoskeleton and in modulating the severity or progression of glomerular disease. To test this hypothesis, I propose the following three Specific Aims: 1.Elucidate the functional roles of synaptopodin in podocytes in the animal. 2. Generate synaptopodin-deficient podocyte cell lines and test whether the lack of synaptopodin affects the structure, the adhesive properties and the mechanical stability of these cells. 3. Identify and characterize functional **domains** of synaptopodin and interacting proteins. These studies should reveal whether synaptopodin functions in **stabilization** of the actin cytoskeleton and in providing podocyte resistance to strain and whether synpo-!- mice may be prone to the development of glomerular injury.

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TI Molecular and Cellular Mechanisms in Agriculture: Structure/Function Relationships of Agriculturally Relevant Macromolecules

SUM To understand the relationship between structure and function in the macromolecules that perform catalytic and other essectial activities in the cell. The macromolecules and macromolecular structures that are the focus of these studies are bacterial ribosomes, plant proteins (leghemoglobins, photosystem I), and animal proteins (Tec protein kinase, glucoamylase, hexokinase, fructose-1,6-bisphosphatase, and dextran sucrase). These studies investigate the mechanisms of enzyme catalysis (glucoamylase, hexokinase, and fructose-1,6-bisphosphatase), enzyme regulation (Tec protein kinase), enzyme and protein in interactions will small gaseous molecules or with electrons (leghemoglobin, photosystem I), oxidative effects (animal proteins) and protein interactions with nucleic acids (ribosomes). Once the impact of structure on function is understood for enzymes and motor proteins, the agricultural sciences will have the option of modifying structure in a rational way so as to modify function. This project addresses specific examples of structure/function relationships that may have particular agricultural applications. In addition, the project seeks to discover general principles of protein function that will contribute to the knowledge base from which novel agricultural developments can be made in the future. A wide variety of chemical and physical methods will be employed. In general the approach is to purify specific biomolecules or biomolecular complexes, to determine their three dimensional structure, and from that information to infer how the structure can be translated into a biological function in the in vivo setting. The techniques include: X-ray crystallographic analysis, NMR spectroscopy, EPR spectroscopy, kinetic analsyis of enzyme action, bacterial expression of components and reconstitution of intact macromolecular complexes, protection from chemical modification to determine interfaces between subunits, and molecular genetics to alter structures in living cells and monitor the effects.PR molecular details of T cell activation. NMR spectroscopy is used to elucidate protein structures and protein interactions. This structural information provides the basis for functional assays aimed at understanding the cellular events that lead to a proper immune response. This interdisciplinary approach has yielded precise information regarding the molecular mechanisms that control an organism?s response to infection. 2) The Fromm **group** is focused on structure function relationships associated with enzymes of carbohydrate metabolism and purine nucleotide biosynthesis. Using techniques involving enzymology, molecular biology, and x-ray diffraction crystallography, they have established the catalytic mechanisms and mechanisms of molecular regulation of a number of enzyme systems. 3) Dr. Thomas? primary focus this last year has been establishing that protein sulfhydryls are irreversibly damaged by oxidation in intact animals, and that this damage is increased in aging. We have examined heart, skeletal muscle and liver tissue and found that damage is increased approximately 3

fold in aged animals. This damage has also been demonstrated in human hemoglobin from aged individuals. 4) The Hargrove laboratory is interested in the structure and function of heme proteins. Structural mechanisms which regulate ligand binding are investigated using site directed mutagenesis, spectroscopic, equilibrium, and kinetic methods in combination with X-ray crystallography. A significant focus of my laboratory is on the structure and function of a diverse family of proteins called hexacoordinate hemoglobins found in many organisms including bacteria, plants, and animals. Hexacoordinate hemoglobins are thought to serve a novel, common function in both plants and animals. A second major research area are the general mechanisms of regulation of ligand binding and heme iron reduction in hemoglobins. 5) Ribonucleoprotein particles (RNP) play essential roles in virtually all aspects of cell growth and regulation. The focus of Dr. Culver?s research is to understand the dynamic assembly and function of these particles. Primarily efforts are focused on understanding assembly of the ribosome a highly complex RNP responsible for all cellular protein synthesis. To date we have identified RNA conformational changes that are critical for assembly and have, for the first time, identified factors that facilitate this process. 6) A new spin labeling EPR approach is used In Professor Shin's laboratory to study the structure and function of biologically important membrane proteins. The strategy is to site-specifically place a nitroxide spin label in the protein by replacing a native residue with cysteine, which provides a unique labeling site. Current developments of EPR technology make it possible to obtain information on secondary and tertiary structures as well as membrane topology using spin labeled mutants.PB characterization of a **proline**-driven conformation switch within the Itk SH2 **domain**. Nature Structural Biology 9:900-905.PB structures of tachyplesin I and its active linear derivatives. Biochemistry 41:12359?12368.PB tyrosine kinase Itk by the peptidyl-prolyl isomerase cyclophilin A. Proc. Natl. Acad. Sci. USA 99:1899-1904.PB Andreotti. 2002. Competing modes of self-association in the regulatory **domains** of Bruton?s tyrosine kinase: intramolecular contact vs. asymmetric homodimerization. Protein Science 11:36-45.PB active site of Adenylosuccinate synthetase from Eschericha coli. J. Biol. Chemical 277:5970-5976.PB Determinants of L-aspartate and IMP Recognition in Escherichia coli Adenylosuccinate Synthetase. J. Biol. Chemical 277:8817-8821.PB Liver Fructose-1,6-bisphosphatase Reveal Multiple Pathways of Allosteric Inhibition. J. Biol. Chemical, 277:15539-15545.PB 6-phosphoryl-IMP Complexes of Recombinant Mouse-muscle Adenylosuccinate Synthetase. J. Biol. Chemical 277:26779-26787.PB and Product Complexes of Recombinant Mouse-muscle Adenylosuccinate Synthetase. J. Biol. Chemical 277:40536-40543.PB Irreversible Thiol oxidation in Carbonic Anhydrase III: Protection by S-glutathiolation and Detection in Aging Rats. Biol.Chemical 383:649-662.PB Quantitation of Protein Sulfinic and Sulfonic Acid, Irreversibly Oxidized Protein Cysteine Sites in Cellular Proteins. In: Methods in Enzymology, 'Protein Sensors of Reactive Oxygen Species: Selenoproteins, **Thioredoxin**, Thiol Enzymes and Proteins.' Ed. Lester Packer and Helmut Sies, Academic Press, Inc., Orlando FL., 348:146-156.PB hexacoordinate hemoglobin. J. Biol Chemical 277:19538-19545.PB Hargrove. 2002. The leghemoglobin proximal heme pocket directs oxygen dissociation and **stabilizes** bound heme. Proteins. 46:268-277.PB facilitates 30S ribosomal subunit assembly. Mol Cell. 10:129-138.PB with a U3 SnoRNP. Chem Biol. 9:777-779.PB complex. Biochemistry. 41:9264-9268.PB bundle of the neuronal t-SNARE complex is neither disordered in the middle nor uncoiled at the C-terminal region. J. Biol. Chemical 277:24294-24298.PB **domain** swapping for the neuronal SNARE complex with electron paramagnetic resonance. Biochemistry 41:5449-5452.CA

L99 ANSWER 67 OF 71 GENBANK® COPYRIGHT 2004 on STN

JOURNAL (SO): Proc. Natl. Acad. Sci. U.S.A., 100 (14), 8298-8303 (2003)

JOURNAL (SO): Submitted (21-JAN-2003) Max Planck Institute for Molecular Genetics, proScience Ihnestrasse 73, D-14195 Berlin, Germany Max Planck Institute for Marine

Microbiology Celsiusstrasse 1, D-28359 Bremen, Germany

AUTHOR (AU): Gloeckner,F.O.; Kube,M.; Bauer,M.; Teeling,H.; Lombardot,T.; Ludwig,W.; Gade,D.; Beck,A.; Borzym,K.; Heitmann,K.; Rabus,R.; Schlesner,H.; Amann,R.; Reinhardt,R.

AUTHOR (AU): Kube,M.; Borzym,K.; Heitmann,K.; Klages,S.; Marquardt,I.; Lehrack,S.; Beck,A.; Pawlik,R.; Reinhardt,R.; Gloeckner,F.O.; Bauer,M.; Teeling,H.; Lombardot,T.; Ludwig,W.; Gade,D.; Rabus,R.; Schlesner,H.; Amann,R.

TITLE (TI): Complete genome sequence of the marine planctomycete Pirellula sp. strain 1

TITLE (TI): Direct Submission

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Reinhardt,R.

AUTHOR (AU): Kube,M.; Borzym,K.; Heitmann,K.; Klages,S.;
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TITLE (TI): Direct Submission

L99 ANSWER 71 OF 71 GENBANK® COPYRIGHT 2004 on STN

JOURNAL (SO): Nature, 417 (6885), 141-147 (2002)

JOURNAL (SO): Submitted (09-MAY-2002) Submitted on behalf of the
Streptomyces sequencing team, Sanger Institute,
Wellcome Trust Genome Campus, Hinxton, Cambridge CB10
1SA E-mail: sdb@sanger.ac.uk

AUTHOR (AU): Bentley,S.D.; Chater,K.F.; Cerdeno-Tarraga,A.M.;
Challis,G.L.; Thomson,N.R.; James,K.D.; Harris,D.E.;
Quail,M.A.; Kieser,H.; Harper,D.; Bateman,A.; Brown,S.;
Chandra,G.; Chen,C.W.; Collins,M.; Cronin,A.;
Fraser,A.; Goble,A.; Hidalgo,J.; Hornsby,T.;
Howarth,S.; Huang,C.H.; Kieser,T.; Larke,L.; Murphy,L.;
Oliver,K.; O'Neil,S.; Rabbinowitsch,E.;
Rajandream,M.A.; Rutherford,K.; Rutter,S.; Seeger,K.;
Saunders,D.; Sharp,S.; Squares,R.; Squares,S.;
Taylor,K.; Warren,T.; Wietzorrek,A.; Woodward,J.;
Barrell,B.G.; Parkhill,J.; Hopwood,D.A.

AUTHOR (AU): Bentley,S.D.

TITLE (TI): Complete genome sequence of the model actinomycete
Streptomyces coelicolor A3(2)

TITLE (TI): Direct Submission

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